ENVIRONMENTAL TOBACCO SMOKE

A REVIEW OF THE LITERATURE

2001457377

October, 1988

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

TABLE OF CONTENTS

INTRODUCTION..... 1

ENVIRONMENTAL TOBACCO SMOKE AND HEALTH CLAIMS..... 5

An Overview..... 5
Lung Cancer..... 9
Lung Function/Disease: Adults..... 21
"Compromised" Individuals..... 25
Heart Disease..... 27
"Compromised" Individuals..... 28
Lung Function/Disease: Children..... 30
Allergy..... 34
Sick Building Syndrome..... 36

ENVIRONMENTAL TOBACCO SMOKE AND INDOOR AIR QUALITY..... 39

Sidestream Smoke..... 39
Constituents of ETS..... 42
Carbon Monoxide..... 42
Particulates..... 44
Nicotine..... 46
Nitrosamines..... 49
Other Constituents..... 50
Cotinine as a Marker for ETS Exposure..... 51
Summary..... 52

ENVIRONMENTAL TOBACCO SMOKE AND THE PUBLIC..... 53

Government Regulations and Individual Rights..... 53
What is the Solution?..... 60

REFERENCES..... 61

2501457400

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

INTRODUCTION

The claim that exposure to environmental tobacco smoke (ETS) is harmful to the health of nonsmokers has generated a great deal of public concern. Although a review of the scientific literature indicates that this claim has not been proved, it is used by antismokers in their efforts to make smoking socially unacceptable and, thereby, to create a "smoke-free" society. The ETS/health issue is so critical to the antismoking movement that the Director of the U.S. Office of Smoking and Health has remarked that "of all the issues, this [ETS] is the one that will propel the United States toward a smoke-free society."[1]

According to one sociologist, the program for a "smoke-free" society may be characterized as follows:

> The strategy, quite overtly, is to progressively stigmatize smoking, segregating the smoker in all public places, and eventually to eliminate smoking as a socially acceptable custom. How is this to be made politically palatable? The answer is clear: by suggesting that smoking harms not only the smoker, but various categories of "innocent bystanders."[2]

The questionable and often contradictory nature of studies concerning ETS exposure and health is largely ignored by the antismoker; the goal is to achieve publicity and arouse emotion. This strategy is typified by a statement from a commentator on the proceedings of the Fifth World Conference on Smoking

2501457401

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

and Health: "Regardless of the ultimate validity of the findings, which remains to be established, studies such as these have brought the issue of passive smoking to the public's attention."[3]

In addition, the issue of nonsmoker exposure to ETS has become the means by which antismokers seek to enact legislation regulating smoking in the workplace and other public places. Here again, the strategy of using the ETS/health argument to modify smoking behavior is apparent. For example, the chairperson of a U.S. National Academy of Sciences committee which authored a recent report on ETS remarked that "legislation could also be justified, not because it will have much effect on the occurrence of lung cancer among nonsmokers, but because it may motivate some people either to stop smoking or never to start."[4] Indeed, one reviewer observed that there are even instances in the scientific literature in which calls are made to establish ETS as harmful "as a means to limit smoking."[5]

However, the tactic of using the ETS/health argument to modify smoking behavior has been criticized in the international press. One columnist for The (London) Times noted that while she agreed in principle with the antismoking movement, she could not condone "the lying that accompanies it." She explained: "The lie is the claim that the health hazards of second-hand smoke have been scientifically established."[6] Similarly, a writer for a German publication called the use of the ETS/health argument an

2501457402

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

"unethical" tactic intended to instill "anxiety in the nonsmoker" and "feelings of guilt in the smoker."[7]

Certainly, ignorance of scientific procedures and the inability to recognize faulty research methodology play a role in the controversy on ETS. As one reviewer of the literature wrote in 1987:[8]

> A pluralistic society such as ours has lobbies. And it is they who have removed the subject of passive smoking from the scientific to the political stage. It would be wrong to input this to bad intentions. Inadequate or biassed occupation with the problem or general insecurity with regard to scientific comprehension, are quite enough.

Indeed, in another recent review article, a German scientist, Dr. K. Uberla, cautioned that the argument that ETS causes lung cancer in nonsmokers may "have serious negative effects on the credibility of epidemiology" and may become an "obstacle" to the antismokers' "goal" of the reduction of active smoking.[9] Dr. Uberla based his observation upon a comprehensive review of the studies to date on ETS and lung cancer. He concluded that, at best, the data for establishing an association are "inadequate" and that, consequently, "a causal connection cannot be inferred."

Given the importance of the ETS issue for the antismoking movement, the purpose of this paper is to review the current scientific literature regarding environmental tobacco smoke and the health of nonsmokers. It is intended to identify sources of

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

information and misinformation about ETS in an effort to demonstrate that the weight of the scientific evidence does not justify the claim that ETS causes disease in nonsmokers.

2501457404

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

ENVIRONMENTAL TOBACCO SMOKE AND HEALTH CLAIMS

An Overview

Antismokers typically refer to several highly publicized studies as "proof" that tobacco smoke is harmful to nonsmokers. The studies most frequently cited are those by Hirayama[10] and Trichopoulos, et al. (1981)[11], who reported that nonsmokers exposed to tobacco smoke have a higher risk of developing lung cancer, and White and Froeb (1980)[12], who asserted that nonsmokers with long-term exposure to tobacco smoke in the workplace have significantly reduced small-airways function. Still other reports have suggested that children and individuals with lung and heart disease are adversely affected by exposure to environmental tobacco smoke.

In addition, government health officials have issued reports which antismokers are using to pressure legislative bodies in the promotion of a "smoke-free" society. In 1986, two such reports in the U.S., one issued by a National Academy of Sciences (NAS) committee to the U.S. Environmental Protection Agency[13], and the other by the Office of the Surgeon General[14], concluded that ETS exposure is harmful to nonsmokers. More recently, the Independent Scientific Committee on Smoking and Health in the United Kingdom issued its report with conclusions similar to those of the U.S. Surgeon General.[15]

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

However, critical analyses of these studies and reports by physicians and scientists from around the world, together with other studies which reported finding no association between ETS and human disease, show that such adverse health claims are highly suspect and not justified on a scientific basis. For example, in 1983, under the sponsorship of the U.S. Tobacco Institute, an international group of scientists and physicians met at the University of Geneva to discuss scientific developments relating to ETS. The organizer of the symposium summarized their discussions as follows:

> An overall evaluation based upon available scientific data leads to the conclusion that an increased risk [of lung cancer] for nonsmokers from ETS exposure has not been established.[16]

In May of 1983, scientists and epidemiologists who attended a workshop on "Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies" sponsored by the U.S. National Institutes of Health concluded:

> A review of the data from the studies which have been carried out or are in progress which address the effect of passive smoking on the respiratory system suggest that the effect varies from negligible to quite small.[17]

Similarly, organizers of a symposium on the "Medical Perspectives on Passive Smoking" held in Vienna in April, 1984 and co-sponsored by the German and Austrian societies for industrial

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

medicine in cooperation with the International Green Cross, concluded:

> Should lawmakers wish to take legislative measures with regard to environmental tobacco smoke, they will, for the present, not be able to base their efforts on a demonstrated health hazard from environmental tobacco smoke.[18]

Indeed, in the preface to the 1986 reprint of those proceedings published by the International Green Cross, Dr. Joseph Handler, a former Director of the World Health Organization (WHO), observed that "the question as to whether the conceivable theoretical possibility of a risk [from ETS] calls for preventive measures is not a medical but rather a politico-social one."[19]

In October, 1986, an international gathering of scientists in Essen, Germany, considered the experimental and toxicological findings regarding ETS.[20] Professor J.G. Gostomzyk, director of the Health Bureau in Augsburg, Germany, concluded in his review of the proceedings that "so far, even toxicology has not been able to ascertain with any greater degree of probability than did epidemiology that there exists a link between damage to health and passive smoking."[21]

At the conclusion of the International Conference on Indoor Air Quality held in Tokyo, Japan, in November 1987, the chairman of the organizing committee called for more research on the ETS issue, while cautioning against the use of restrictive measures prior to establishing an adequate scientific basis.[22]

2501457407

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

In May of 1988, organizers of a symposium on ETS in Austria entitled "Illness Due to Passive Smoking?" issued a press release in which they concluded that "a causal relationship between ETS and illness cannot be established" and that "there is no positive evidence that cancer and other such illnesses are caused by passive smoking."[23]

Other scientists recently have reached similar conclusions. Dr. Paul Witorsch, Professor of Medicine at George Washington University in Washington, D.C., concluded that "a thorough and critical examination of the relevant literature fails to provide compelling evidence that exposure to ambient tobacco smoke produces adverse chronic health effects."[24] Similarly, Dr. Domingo Aviado, a physician and former professor of pharmacology, wrote that his review of the literature "indicates that there is no substantial evidence to support the view that exposure to environmental tobacco smoke presents a significant health hazard to the nonsmoker."[25]

Moreover, in a study published in 1986, researchers in the United Kingdom who had investigated what impact ETS has on the health of nonsmokers concluded: "Amongst lifelong non-smokers, passive smoking was not associated with any significant increase in risk of lung cancer, chronic bronchitis, ischaemic heart disease or stroke in any analysis."[26]

Given such findings, it appears remarkable that anti-smokers continue to claim that exposure to environmental tobacco

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

smoke is a proven cause of disease in nonsmokers. Obviously, this claim carries a great deal of emotional weight even without appropriate scientific support. Therefore, the charges are serious enough to warrant close examination.

Lung Cancer

Two highly publicized articles published in 1981 are still among the most frequently cited to support the claim that ETS exposure increases the nonsmoker's risk of lung cancer. A study of Japanese women by Dr. Takeshi Hirayama reported that nonsmoking wives of heavy smokers have a much greater risk of developing lung cancer than nonsmoking wives of nonsmokers.[10] In a study of Greek women, Trichopoulos, et al., concluded that a nonsmoking woman whose husband smokes has twice the risk of developing lung cancer as a nonsmoking woman married to a nonsmoker.[11]

Both studies have been widely criticized in the scientific literature. Numerous inadequacies and inconsistencies in the Hirayama study have been pointed out by noted scientists and physicians who have questioned the validity of its conclusions.[8,27-43] Dr. Ragnar Rylander, the organizer of the 1983 University of Geneva symposium on ETS, noted that the study had been criticized for its lack of questionnaire reliability, absence of histological diagnosis, questionable statistical treatment, and

2501457409

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

failure to examine such factors as air pollution from heating and cooking.[44] A participant at the International Conference on Indoor Air Quality held in Tokyo in 1987 noted, in a published report, that several recent critiques had questioned Hirayama's study for bias in statistical processing, as well as for failures to measure ETS exposure levels and to evaluate and report "dietary and occupational factors known to influence susceptibility to lung cancer."[27] Moreover, two studies presented at the Conference on Indoor and Ambient Air Quality, held at Imperial College in London in 1988, reported that Hirayama had not divided his study population into appropriate age groups. The presenters noted that the risk he reported for nonsmoking wives of smokers disappears when such age bias is removed.[28,33]

Criticisms of the Trichopoulos, et al., study were acknowledged by the authors in the 1983 update of the study.[45] They noted that their research had been "criticized (by ourselves and others) because of the small number of subjects, because several tumors lacked histological confirmation, and because controls and cases were from different hospitals."

Moreover, their findings were inconsistent with another study published that year. In late 1981, Lawrence Garfinkel, an official with the American Cancer Society (ACS), reported the results from a follow-up of the group's long-term lifestyle study, involving nearly 180,000 nonsmoking American women divided into categories based on the amount their husbands smoked.[46] By

2501457410

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

comparing the lung cancer mortality rates of women reportedly exposed to different levels of tobacco smoke, he determined that none of the differences observed were statistically significant and that "compared to nonsmoking women married to nonsmoking husbands, nonsmokers married to smoking husbands showed very little, if any, increased risk of lung cancer." Moreover, Garfinkel cautioned that any study based solely upon classifications of the husbands' smoking habits could not account for total exposure, and that, therefore, the results could be misleading. (See also Garfinkel et al., 1985.)

Several subsequent studies have claimed to support the reported findings of Hirayama and Trichopoulos, et al.,[47-52,54] but a close examination of the methods and conclusions of these studies reveals that their claims are scientifically unfounded. For example, a report by Knoth, et al., which identified 39 nonsmoker lung cancer cases, asserted that exposure to ETS was the "most plausible explanation" for the reported lung cancers.[52] However, the study failed to provide a comparison group as a control population. One reviewer consequently characterized the report as containing "only tentative conclusions based on poor data analyzed by unacceptable methods."[53]

In a 1983 study, Correa, et al., reported an increased risk of lung cancer for nonsmokers married to smokers in a southern U.S. state.[54] However, the results were based on only 30 cases and did not take into account occupational, indoor and outdoor

2501457411

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

exposures or other confounding variables. Furthermore, one of the co-authors of this study later issued a report with contradictory findings for a similar population in a neighboring state.[55]

In 1985, a published article by two antismoking advocates, Repace and Lowrey, estimated that exposure to ETS is responsible for 500 to 5,000 lung cancer deaths a year among nonsmokers in the United States.[56] The risk estimates were cited widely in the press prior to the report's publication, perhaps because it was incorrectly identified as a study by the U.S. Environmental Protection Agency (EPA). In reality, the report was neither approved nor endorsed by that agency. Indeed, Repace now faces a full-scale ethics inquiry into charges that he was paid for his involvement in antismoking activities while he was an EPA employee.[57]

The report itself presents two highly theoretical models for estimating risks of lung cancer from ETS exposures. The first model relies upon a "reinterpretation" of the epidemiological studies of lung cancer in nonsmokers, and the second provides an estimate for lung cancer mortality from a single study dealing with the Seventh Day Adventists, a religious group known for its vigorous opposition to smoking.

Critics of this approach have pointed out that the estimates are based on errors and "unrealistic assumptions" which result in overestimations of exposure.[58,59] For example, one recent analysis of their model showed that, depending upon the

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

assumptions and input data used, such model estimates are inherently inaccurate and may vary by as much as 300-fold.[60] Another scientist noted that the exposure and dose levels they used are not based on actual measurements, and that actual measurements reported by other researchers range from "ten-to-one-hundred-fold less than that in the Repace and Lowrey model."[61] Still other scientists have questioned the methods of analysis used in their report.[62-64] They suggest that Repace and Lowrey failed to control for other confounding factors, and that their model did not provide "the very statistical bases of estimation procedures."[61]

Repace and Lowrey's estimate of nonsmoker lung cancer risks in the workplace has been criticized by another group of scientists who pointed out that none of the epidemiological studies which examine the relationship between ETS exposure and disease in nonsmoking working women reports a statistically significant increase in risk.[58]

Additional studies have claimed that the risk for all cancers is increased among nonsmokers exposed to ETS. One such study examined a population which was selected from death notices in a northeastern U.S. newspaper.[65] However, analysis of the data reveals that the reported risk disappears when age comparisons of cases and controls are considered.[66]

In a series of papers, Sandler, et al., reported increased risks for lung cancer and cancer of other sites in nonsmokers exposed to ETS.[67-69] Yet published criticisms cast seri-

2501457413

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

ous doubt on these conclusions. One commentator challenged the authors' selection of controls and their use of questionnaire information.[70] Another scientist observed that under one interpretation, the data suggest that exposure to ETS had no effect on overall cancer incidence.[71-72] A third critic called the studies "seriously flawed" and questioned the authors' methods of data collection.[73] Still others challenged their conclusions because the reports had not considered potential confounding variables such as diet, hygiene and lifestyle.[74-75]

With regard to the issue of ETS and overall cancer mortality, a 25-year follow-up study of over 1,000 married couples in Holland reported that "passive smoking was not associated with an increase in total mortality."[76] Although the authors cautioned that the results do not necessarily contradict those which suggest such an association, they added that their findings "were reassuring to the extent that the possible absolute risk carried by passive smoking is probably small."

The complex nature of the scientific data in this area is further illustrated by the mixed results of some studies; that is, certain data in a study may suggest a relationship between ETS and lung cancer while other data from the same study may not. For example, in a report on the preliminary results of a health survey conducted in two urban communities in Scotland, Gillis, et al., noted that of the six lung cancer deaths observed in male nonsmokers, four were among those who had reported expo-

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

sure to ETS.[77] However, no dose-response relationship was noted for females in the study.

Kabat and Wynder, in a 1984 study of 25 male and 53 female nonsmoker lung cancer cases, reported a greater amount of ETS exposure at work for male nonsmoking cases.[78] However, no significant effect was reported for males exposed to ETS at home or for female cases exposed either at work or at home.

In 1985, Garfinkel, et al., published a study of 134 nonsmoking women with lung cancer who were selected from hospital records from 1971 to 1981.[79] Garfinkel, et al., provided two analyses of the same data set which produced apparently contradictory results. While a statistically significant dose-response relationship was reported between nonsmoking female lung cancer patients and the number of cigarettes smoked per day by their spouses, no significant relationship was observed between the occurrence of lung cancer in nonsmoking women and the total number of hours per day of exposure to ETS over the last five or 25 years.

Still other studies have observed no significant association between ETS and lung cancer. These include a 1982 study from Hong Kong which reported fewer "passive smokers" among lung cancer patients than among controls.[80] The authors noted that "this finding is at variance with that of Dr. Hirayama's."

Similarly, Koo, et al., studied 200 female lung cancer cases among Chinese women and concluded that ETS exposure in the home or at work had no statistically significant impact on lung

2501457415

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

cancer incidence.[81] In a second study published in 1987, Koo et al., calculated total ETS exposures at home and at work for their population of nonsmoking female patients. They observed no significant disease trends for any of the lifetime measurements of exposure to ETS.[82]

In two papers published in 1988, Dr. Koo re-analyzed her data set on nonsmoking wives of smokers.[83,84] Her results indicated that wives of nonsmoking husbands had "healthier" lifestyles than wives with smoking husbands; they exhibited higher socio-economic status and had better indices of family cohesiveness and lower frequencies of selected health problems and complaints. An important and statistically significant difference was found in the diets of the two groups. Wives with smoking husbands consumed more processed and spicy foods and ate fewer fresh fruits and vegetables than did wives of nonsmoking men. Koo concluded that such correlates of smoking status "act as important confounders when evaluating health risks among families with smoking husbands."

In 1985, Wu, et al., reported on data from a study of female lung cancer cases in a large metropolitan area in the U.S.[85] They reported that the lung cancer risk among the 31 nonsmokers in the study population was not affected by ETS exposure.

Researchers have recently published results of a study undertaken to investigate the occurrence of lung cancer in an industrial area in Scotland.[86] The authors reported that ETS exposures were not significantly different between the cases and

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

controls in the study; in other words, ETS appeared to play no role in the reported increased incidence of lung cancer in the study population.

In 1986, researchers investigated the potential role of ETS on the incidence of nonsmoker lung cancer in England.[26] The researchers observed no significant trends in increased risk for lung cancer patients who reported exposure to ETS. In fact, in some disease categories, more controls than cases reported exposure to ETS.

Perhaps as a result of the failure of individual studies to report consistently significant results, certain researchers have recently begun using another method of statistical analysis which combines the reported results from numerous studies. With this method, called "meta-analysis", they claim to have calculated estimated excess risks for nonsmokers exposed to ETS that are 10 to 50 percent greater than those for nonexposed nonsmokers. In basic terms, they have reached these broad conclusions or "generalizations" by calculating an average of the relative risks for nonsmoker lung cancer cases reported by those studies.[15,87-89]

However, this method of generalization has been criticized for its questionable application to those studies, due to the wide variety in their study designs, population selections, techniques of analysis and their results.[90-92] This method also ignores basic methodologic weaknesses which, according to one recent

2501457417

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

governmental report, are characteristic of each published study on ETS and lung cancer.[93] It further ignores the fact that the majority of studies considered in the various "meta-analyses" do not report statistically significant increases in lung cancer risks for nonsmokers. For example, only two of the thirteen studies considered by the 1986 NAS Report achieved statistical significance.[94] (Although the authors of several studies report no statistically significant risks, they report "positive" trends in their data. However, trends which fail the test for statistical significance are of questionable scientific validity).

In a recent analysis, Letzel and co-workers considered only those epidemiological studies on ETS which met minimum requirements of scientific quality. The researchers then performed their own meta-analysis on those studies with corrections for misclassification (see discussion below). They concluded that the computed excess risk was negligible and not statistically significant.[92]

Peter Lee, a statistician and epidemiologist from the United Kingdom, has argued that the increased risks reported in various "meta-analyses" are the result of an inherent bias in study design rather than the result of any genuine effect from exposure to ETS.[95-100] Lee presents data which indicate that the reported risks cannot be explained on the basis of either ETS exposure or dose for the nonsmoker. It is Lee's contention that the reported

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

"risks" are the result of bias caused by a small number of smokers who are misreported in the studies as nonsmokers.

Other kinds of misclassification may contribute to the reported increase in lung cancer risks among nonsmokers, according to several scientists. For example, none of the studies on ETS and lung cancer provides direct observational information on ETS exposures. Instead, spouses, next-of-kin or friends are asked to estimate the amount of ETS to which they think the subject was exposed. Such estimates may lead to a kind of misclassification, called exposure misclassification,[101] which has been shown by Garfinkel[79] and Friedman[102] and others[103-106] to lead to improper indices of exposure and incorrect estimations of risk. In Garfinkel's study, for example, relative risks varied from 0.83 and 0.77 when the women with lung cancer or the husband was the respondent, to a risk of 3.57 when a son or daughter responded.[91]

Dr. S. James Kilpatrick, a biostatistician from the Medical College of Virginia, has analyzed another form of misclassification, called differential misclassification, which results "from the tendency of respondents to inflate the amount of ETS exposure for lung cancer cases and deflate the report of exposure for controls."[101] Similarly, Dr. Ernst Wynder, President of the American Health Foundation, notes that "relatives of a nonsmoking lung cancer patient are more likely to report passive inhalation exposure on the part of their relative than are relatives of a control patient."[107]

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

A more subtle form of potential bias is known as "publication bias", which is generated by the failure to publish studies which report negative or weakly positive results.[108,109] Scientists have recently expressed concern over the growing trend in the literature which tends to overemphasize (and hence publish) only those studies which report increases in risk.[110,111] Published studies which are thus combined for meta-analyses may not truly represent all investigations on the topic of ETS exposures and lung cancer.

Most of the epidemiological studies on ETS and lung cancer have failed to consider age differences, diet, occupation and exposures to indoor or outdoor pollution as potential confounding elements. The importance of such factors is underscored by four recently published reports, one from Japan and three from China.[112-115] The reports suggest that indoor pollution generated by kerosene heaters, coal stoves, liquified petroleum gas and exposures to cooking oil vapors may be responsible for the increased risk of lung cancer among Oriental women. In addition, relatively few studies have considered ETS exposures outside the home; only four studies sought information about total exposure to ETS from various sources.[26,79,81,82] Interestingly, none of these studies report a significant association between total ETS exposure and lung cancer.

Given these difficulties in interpretation, it is therefore not surprising that an eminent statistician should

2501457420

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

conclude that "it is unlikely that any epidemiological study has been, or can be, conducted which could permit establishing that the risk of lung cancer has been raised by passive smoking. Whether or not the risk is raised remains to be taken as a matter of faith according to one's choice."[108]

Lung Function/Disease: Adults

The issue of ETS and respiratory disease in nonsmokers was raised in a 1980 study by White and Froeb.[12] They measured the small-airways function of smokers and nonsmokers and concluded that nonsmokers exposed to tobacco smoke at work for 20 or more years had reduced function of the small airways compared to nonsmokers not so exposed. Similarly, in 1983, French researchers reported that nonsmoking spouses over 40 years of age who were married to smokers exhibited decreases in pulmonary function compared to nonsmoking spouses of nonsmokers.[116]

The White and Froeb study has been criticized for a number of reasons.[117-119] For example, a physician at a U.S. medical school questioned the authors' use of carbon monoxide as an index of smoke exposure, contending that they "do not have reliable estimates of the smoke exposure in the environment of their nonsmokers" because it is not unique to tobacco smoke.[120]

Based upon his own analysis of the White and Froeb study, a British reviewer concluded that the authors' findings

2501457421

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

"relate to an index which is contentious and certainly not an accepted reliable indicator of an increased health risk."[34] White and Froeb themselves noted that the average values of the pulmonary tests of nonsmokers exposed to tobacco smoke "were not notably different" from the values suggested as normal by a specialist in this area.[121]

Perhaps the most telling criticism of the study was voiced by Dr. Michael Lebowitz of the University of Arizona at an annual joint meeting of the American Lung Association/American Thoracic Society and in a subsequent letter published in the U.S. Congressional Record.[122-123] During a forum at the ALA meeting, Dr. Lebowitz stated that he had concluded, from his own extensive review of the study and from an interview with White, that the study was "improperly designed" from an epidemiological point of view. He said that there were problems "inherent in the study," including the selection of the study group and the proper measurement of smoke in the workplace. Dr. Lebowitz also expressed concern that the statistical significance of the data appeared to depend on the unexplained omission of data for 3,000 people who were originally included in the study. Based upon these considerations, Dr. Lebowitz urged that the study not be used to support the claim that environmental tobacco smoke affects the lung function of adults in the workplace.

In a paper presented to the 1984 Vienna Symposium,

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Dr. Lebowitz again took issue with the White and Froeb study. He wrote:

> Even with a biased population, poor study design, and incorrect statistical evaluation, there were no clear-cut, consistent, medically meaningful differences between passive smokers and groups of nonsmokers; a corrected statistical analysis strengthened this conclusion.[124]

Dr. Lebowitz also questioned the finding of the 1983 French study which reportedly showed significant differences in lung function in one part of the study population, although no significant differences between exposed and nonexposed nonsmokers were reported in the population as a whole. Dr. Lebowitz noted that since the "healthiest" population in the study lived in the most polluted areas, the study may have been flawed due to biased population selection and testing or other confounding factors.[124]

In addition, neither the White and Froeb nor the French study is consistent with other research on lung disease and lung function in nonsmokers. For example, a 1984 study of 1,351 German office workers reportedly found no effect on pulmonary function among exposed nonsmokers.[125] In a 1988 update of the study, the investigators noted that "there is no evidence that average everyday passive smoke exposure in the office or at home leads to an essential reduction of lung function in healthy adults."[126] Canadian researchers reported that a group of healthy young adult nonsmokers showed "no consistent reaction" of static lung volumes

2501457423

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

and insignificant changes of other lung function measurements when exposed to tobacco smoke.[127]

Numerous studies of individuals exposed to ETS in the home also present conclusions which are contrary to both the White and Froeb and the French study. In 1981, several epidemiologists at the Johns Hopkins University School of Medicine reported that in a group of 1,724 adults, the frequency of respiratory symptoms in nonsmokers was not associated with the number of smokers in the household.[128] Nor was the frequency of impaired ventilatory function significantly higher if there were smokers in the home. However, their analysis did show that "among men who never smoked cigarettes, gas cooking was definitely associated with impaired ventilatory function, even when corrected for multiple comparisons."

Other studies on pulmonary function, respiratory disease and environmental tobacco smoke deserve special mention. In 1983, for example, Jones, et al., reported that in a case-control study of several hundred nonsmoking women from a U.S. study population, there was no significant association between decreases in lung function and exposure to ETS in the home.[129] Coordinators of an epidemiologic study of obstructive lung disease in Arizona reported finding no effects from ETS exposures in their adult study population.[130]

In a study of 376 families, Yale University scientists reportedly found no evidence that environmental tobacco smoke affected either lung function or symptoms in adults.[131] More

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

recently, in a Canadian study on the effect of indoor and outdoor pollutants on the lung function of housewives, researchers concluded that ETS exposure did not influence lung function but that gas stove usage played a role in average lung function decline.[132]

It is not surprising, therefore, that the participants at the U.S. National Institutes of Health workshop on ETS exposure concluded, after a review of the data from on-going studies on lung function and disease, that a possible effect from environmental tobacco smoke "varies from negligible to quite small."[17]

"Compromised" Individuals: Lung Disease

Many believe asthmatics to be particularly vulnerable to various environmental influences, including ETS. A 1981 study by Dahms, et al., for example, reported decreases in the pulmonary function of several asthmatics exposed to environmental tobacco smoke.[133] However, the study suffers from several obvious limitations. The subjects, for example, were challenged under the unrealistic experimental conditions of exposure to high levels of smoke in an enclosed smog chamber. In addition, as the authors themselves noted, their experiment lacked proper controls and the effects observed may have been due to psychological, not physical, factors.

Contrary to the reported findings of Dahms, et al., a Canadian group who examined the reactions of asthmatics to levels

2501457425

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

of tobacco smoke typically found in public places observed no systematic lung changes after such exposure.[134] Later research by the co-authors of this study supports this conclusion. They reported that respiratory data collected from a group of asthmatic volunteers exposed to tobacco smoke "do not suggest that asthmatic subjects have an unusual sensitivity" to such exposure.[135] Although several volunteers claimed that they experienced wheezing and tightness in the chest due to the exposure, the researchers found that the "physiological data give little support to the concept of a subgroup with particular sensitivity." They noted that these reactions probably were due to the "suggestibility" of the subjects.

The difficulty in determining the impact of psychological responses in such studies is clearly demonstrated by the results of two reports from Australia. Although a 1985 report suggests that short-term ETS exposures are capable of inducing reactions in asthmatics,[136] an earlier study tends to support the theory that psychological reactions may partially explain symptoms observed during such exposures. In the latter study, Ing, et al., reported that while asthmatics exposed to tobacco smoke complained of subjective symptoms, no significant objective evidence of airways obstruction was observed.[137]

Another report on results from a large-scale epidemiological study in the U.S. suggests that environmental tobacco smoke in the home does not affect symptoms or pulmonary function in either

2501457426

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

children or adult asthmatics, although dust and pollen in the home apparently can provoke such effects.[138]

A group of researchers from Yale University, in a recently published study on this issue, studied the short-term effects of ETS exposure on a group of young asthmatic patients.[139] They observed no changes in lung flow rates and concluded that such exposures present "no acute respiratory risk" to asthmatics.

Heart Disease

Epidemiologic studies assessing ETS and the risk of cardiovascular disease which appear in the literature show no clear trends. Although two studies reported a small, but statistically significant, increase in heart disease risk among nonsmoking wives of smokers,[140,141] another generated both statistically significant and insignificant results.[142] A fourth study reported a statistically insignificant increased risk,[143] and two additional reported no significant association between heart disease and ETS exposures.[26,77]

In 1984, two German researchers reviewed the available studies on the roles of carbon monoxide (CO), nicotine and other tobacco smoke components in the possible etiology of coronary heart disease in nonsmokers. The authors concluded that "there is little evidence" to suggest that substances found in ETS "may adversely affect the cardiovascular system." They also stated that "neither

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

CO nor nicotine is likely to play a role in the development and progression of coronary heart disease in those concentrations normally found in passive smokers."[144] In addition, scientists and physicians who examined the issue of carbon monoxide at the 1983 Geneva Symposium on ETS agreed that "carbon monoxide from environmental tobacco smoke is not important from a health point of view."[16]

"Compromised" Individuals: Heart Disease

The issue of ETS exposure for individuals with pre-existing heart disease arises primarily from a 1978 study by Aronow who concluded, on the basis of examining ten heart patients, that such exposure hastens the onset of heart pain during exercise.[145]

However, the Aronow experiment has been repeatedly and severely criticized by a number of authorities, including the U.S. Surgeon General.[146] Dr. Edwin Fisher, a professor of pathology at the University of Pittsburgh, stated that the study must be "evaluated in light of the fact that the end-point of the study was highly subjective, that the stress factor was not controlled, and that a sham smoke or other environmental impingement was not used. In other words, not only was the sample small, but the scientific design was exceedingly poor."[147]

After reviewing the Aronow study, a Canadian professor of medicine who otherwise maintains that tobacco smoke exposure

2501457428

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

may be harmful wrote that although the patients showed some increase in carboxyhemoglobin (COHb) or CO blood levels, "the endpoint (the reported onset of anginal pain) is necessarily subjective."[148] He explained, "it is difficult to imagine that enclosure in a very smoky room did not have some emotional impact upon patients who were liable to angina, and the psychological disturbance may have done more to hasten the onset of symptoms than the increase of blood COHb."

In addition, other researchers in the area have been unable to replicate or duplicate Aronow's reported findings. For example, researchers from Great Britain measured objective responses of a group of heart patients to ETS and found no adverse physical response to any level of exposure.[149]

Other serious questions have been raised regarding the underlying validity of Aronow's work. In 1983, after one U.S. governmental agency reported that Aronow had falsified data in a study it had sponsored, another agency, the Environmental Protection Agency (EPA), conducted an independent review of several of Aronow's studies, including his work on heart disease and CO. The EPA concluded that, because of several irregularities in the study, it could no longer rely on his research to formulate environmental standards.[150-151]

2501457429

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Lung Function/Disease: Children

Perhaps no claim regarding ETS is as capable of provoking strong feelings as the charge that parents who smoke may harm the health of their children. While the issue of parental smoking is laden with emotion, the scientific basis for the claim is difficult to interpret. For example, while one study examines respiratory symptoms or illness such as coughs and colds by questionnaire responses from parents,[152] another measures lung function with special equipment at a school or health facility.[153] In the United States alone, according to one report, this has led to a situation in which studies of ETS and the respiratory system are being "carried out by at least three different groups, are employing different populations and methodologies and have led to varying conclusions."[17]

Such studies, each with a different sample size, data collection method and analysis, tend to yield factually incompatible and contrary conclusions. For instance, although certain studies have reported adverse findings,[13,14,154-170] others have observed no significant relationship between parental smoking and respiratory illness in children.[131,152,171-181] After a five-year study of over 400 children, for example, Dutch researchers concluded there was "no evidence" that parental smoking had an appreciable effect on respiratory symptoms in school children.[176] A similar conclusion was reached by a group of U.S. researchers,

2501457430

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

including a critic of smoking, who found "no significant relation" between parental smoking and respiratory symptoms in a study of nearly 400 families with 816 children in three cities.[131]

Moreover, the authors of studies reporting adverse effects from ETS exposures among children concede that their conclusions must be viewed with caution because of numerous confounding factors. For example, one group of British researchers acknowledged the possible influence of factors such as cross-infection in the home and genetic susceptibility to childhood respiratory illness and symptoms.[158,159] More recently, researchers in Hong Kong reported "a highly significant correlation between the frequency of respiratory illnesses" of mothers and their children.[182]

Others have conceded that the reliance on questionnaires for information about respiratory symptoms casts doubt on the findings. In one study which reported a significant association between parental smoking and respiratory symptoms, for example, it was noted that even "slight changes" in the way the questions were phrased could result "in substantial differences in the type of responses one obtains."[160] Similarly, another study observed that there was a significant difference in the respiratory symptoms reported depending on which parent completed the questionnaire.[168]

The importance of such confounding factors was given special consideration in the report from the workshop on ETS

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

sponsored by the U.S. National Institutes of Health.[17] Among the many factors listed in the report were types of heating used, socio-economic status and other variables affecting household conditions, including the number of residents, and demographic and medical characteristics of the study population, such as age, parental symptoms and annoyance responses. The report cautioned "that any study which ignores them will be seriously flawed." The relevance of such factors in affecting the outcome of research on parental smoking is supported by a number of reports which have shown that the use of gas stoves in the home may be independently associated with childhood respiratory disease.[138,174,183-187]

Other confounding factors independent of parental smoking have been reported recently in the medical literature. For example, studies in the United Kingdom have identified damp housing[179,188] and father's occupation[189] as potential explanatory factors for the occurrence of respiratory illness in children. Other recent studies have identified outdoor air pollution,[190,191] infections transmitted during day-care attendance[192] and the use of kerosene heaters in the home[193] as risk factors for childhood respiratory disease.

The contradictory nature of findings on the issue of parental smoking is also apparent in the growing number of studies examining the relationship between parental smoking and children's respiratory or lung function. Although several reports have claimed that parental smoking results in decreased pulmonary lung

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

function in children,[156,160,170,194-200] others have not,[131,201-204] including those of a group of researchers who have published a series of studies on this subject.[130,138,153,205] In 1982, for example, the latter research group showed that a comparison of body size with lung function eliminated any reported correlation between parental smoking and children's lung function.[153] Two years later, a reanalysis of families from their study group again showed that "parental smoking did not have a significant effect on children's pulmonary function; smoking habits of others in the household (predominantly siblings) did not have any effect either."[130]

Thus, claims that parental smoking plays a causal role in the development of respiratory symptoms and reduced lung function in children are not scientifically justified. Such claims are typically based upon a single study of a selected symptom (such as cough or wheeze). These kinds of studies invariably fail to consider nutrition, health habits of the family or other lifestyle variables. Similarly, studies which report reduced lung function in children of smoking parents fail to address the issue of socio-economic status or the potential role of genetic and family traits in pulmonary function capabilities.[204] Moreover, the reductions reported in the literature are small and of uncertain clinical or biological significance, and are contradicted by a number of studies which reportedly have observed no effect of parental smoking on children's lung function.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Allergy

Although some individuals are annoyed by the sight and smell of tobacco smoke and a few even report experiencing irritation, the existence of human allergens in tobacco smoke has not been established scientifically. This is an extremely important point in the ETS debate because those seeking to ban smoking in public places often do so by claiming that nonsmokers are "allergic" to tobacco smoke.

However, a number of research groups have been unable to conclude that a tobacco smoke allergy in humans exists.[206-209] In 1980, for example, a group of researchers noted that "direct evidence that tobacco smoke is immunogenic (capable of evoking a specific response) in man is yet to be documented."[210] A recent report by this same group affirms this conclusion.[211]

Claims about tobacco allergy stem primarily from studies in which tobacco leaf extract has been reported to cause allergic skin responses in some people, usually in those who already experience allergic reactions to other substances such as weeds.[212-214] However, as an English immunologist pointed out, there are "great difficulties" in determining whether positive reactions to tobacco leaf extracts are relevant to clinical responses to tobacco smoke.[209] Although he noted that there may be substances in tobacco smoke which could "theoretically" act as

2501457434

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

such agents, he concluded that "there is no proof that the specific sensitization to tobacco smoke exists." Recent research affirms this point. Scientists reported in 1988 that tobacco leaf sensitivity was not associated with decreased pulmonary function in allergic asthmatics.[215]

It has also been hypothesized that tobacco smoke is capable of provoking asthma as an allergic reaction.[216-217] However, a Swedish specialist concluded that such results are not proof of a tobacco allergy because the studies, which used tobacco extracts, did not differentiate between non-specific and true allergic reactions in evaluating the results of skin tests and bronchial provocation.[218] Consequently, he stated, "for the present, the question as to whether allergy to cigarette smoke exists or not should be kept open."

Certainly, there appear to be people who may be sensitive to tobacco smoke, but personal annoyance and emotional reactions should not be confused with genuine allergic reactions.

In many cases, the individual may be responding to high room temperatures, lack of ventilation, or even to the mere sight of tobacco smoke. Indeed, Dr. John Salvaggio, the director of an allergic disease center in the U.S., has suggested that reported reactions to tobacco smoke may be irritative rather than allergic. After reviewing the studies on the allergy question, he concluded that "there is no proof that tobacco smoke is allergenic in man."[219]

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Accordingly, it is not surprising that researchers at the Mayo Clinic failed to find any evidence of tobacco smoke allergies in their tests of subjects who considered themselves allergic to tobacco and tobacco smoke.[220] It has been pointed out in other studies that people who claim to be "smoke sensitive" did not react more frequently to tobacco leaf or smoke extract than those who are "smoke resistant."[221]

On the basis of these data, it must be questioned what people really mean when they say they are "allergic" to tobacco smoke. They may simply not like the sight and smell of tobacco smoke and interpret this to mean that they are "allergic" to environmental tobacco smoke. But, as has been pointed out, such personal reactions should not be regarded as true tobacco smoke allergies.

Sick Building Syndrome

Advocates of smoking restrictions in the workplace commonly argue that ETS exposure gives rise to a number of complaints, including headaches, nausea, coughs, sore eyes and breathing difficulties. However, recent research indicates that this complex pattern of symptoms, the so-called "sick building syndrome", commonly occurs in modern office buildings whether or not smokers are present.[222-224]

2501457436

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Canadian researchers attending the 1983 University of Geneva symposium on ETS reported on their extensive review of over 150 indoor air quality evaluations of office buildings compiled by U.S. government agencies, universities and others.[223] After an examination of the data, the authors concluded that smoking did not significantly affect either indoor atmospheres or the frequency of worker complaints and symptoms:

> The review of available studies does not provide any objective evidence that either pollution levels or patterns of health related complaints differ in some remarkable way between locations with or without smoking restrictions.

They did observe that "inadequate" ventilation creates conditions "where discomfort and illness result irrespective of whether or not smoking is permitted." The researchers reaffirmed their findings in a report published in 1987.[225]

Similarly, in a review of 203 air quality investigations of government and business offices, schools and health care facilities by the U.S. National Institute of Occupational Safety and Health (NIOSH), U.S. government researchers concluded that tobacco smoke played a contributing role in only four of the buildings investigated.[222] A large majority of all complaints were traced to general building contamination and inadequate ventilation.

In 1988, a representative of a firm specializing in the maintenance of office air conditioning and heating systems reported

2501457437

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

on 223 individual indoor air quality investigations of public and privately owned office buildings.[224] As in the NIOSH investigations, ETS was implicated in only 4 percent of the buildings investigated. He stated that the majority of indoor air quality problems in modern office buildings may be traced to inadequate fresh air circulation and to poorly maintained ventilation systems which act as breeding grounds for fungi, bacteria and other contaminants. He also suggested that visible tobacco smoke ought to be considered a symptom, not a cause, of general indoor air quality problems, in that ETS is often the only visible sign that a ventilation problem exists.

It is therefore understandable, given the easy recognition of ETS, that persons experiencing sick building symptoms may blame ETS as the cause. Indeed, Canadian researchers have verified that the mere visibility or presence of tobacco smoke may provoke claims that ETS is the cause of reported symptoms and complaints.[226]

Thus, not only is ETS erroneously labeled the "cause" of worker discomfort in "building illness" situations, its perceived presence may even heighten anxiety about the safety of an individual's environment. This, of course, adds impetus to the movement for smoking restrictions in the workplace. However, removal of ETS through smoking bans may serve only to divert attention from more basic, underlying indoor air quality problems.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

ENVIRONMENTAL TOBACCO SMOKE AND INDOOR AIR QUALITY

Antismokers often claim that ETS is a major source of indoor pollution in order to support their argument that ETS exposures harm the health of nonsmokers. However, the scientific literature indicates that, except under experimental or other extraordinary conditions, ETS does not have a significant influence on the quality of indoor air.[227]

The claim that ETS is a significant source of indoor air pollution (and, as such, a hazard to health) is based upon several unwarranted assumptions and misrepresentations of the scientific data. This section will provide an analysis of the ETS/indoor air quality question. The nature of ETS, its constituents, its contribution to indoor air, and the methods employed to measure exposure to it will be addressed.

Sidestream Smoke

It is frequently claimed that sidestream smoke, or the smoke from the burning end of the cigarette, contains much higher amounts of certain constituents than mainstream smoke, or the smoke inhaled by smokers. Such a claim implies that a nonsmoker has an increased risk of disease because of his exposure to sidestream smoke, which has more allegedly "toxic" or harmful constituents than smoke inhaled by the smoker. However, this

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

argument is extremely misleading for several reasons, but most notably because it fails to take into account that sidestream smoke is immediately diluted in the surrounding air. This diluted sidestream smoke is more accurately called "ETS".

The impact of dilution on sidestream smoke in room air cannot be overemphasized. Scientists have estimated that under normal, realistic conditions, the quantity of various constituents in ETS are only a tiny fraction--from 1/100th to 1/1000th--of those found in mainstream smoke.[228]

The potential toxicity of ETS was recently assessed in a number of studies.[229-232] These studies typically tested the body fluids of nonsmokers exposed to ETS for mutagens, or substances capable of altering the genetic structure of cells. Although it can be argued that the scientific literature on this subject is very limited, and that the laboratory tests employed are crude, the studies reported no mutagenic activity in the body fluids of non-smokers exposed to ETS.

Animal inhalation experiments using sidestream smoke or constituents of sidestream smoke are also inconclusive.[233-234] German scientists exposed rats and hamsters to very high levels of sidestream smoke during a 90-day inhalation experiment.[233] The researchers reported no significant physiological effects on the tissues of the animals. In his comprehensive review of the literature on suspected pulmonary carcinogens, Dr. Domingo Aviado observed that none of the constituents in sidestream smoke which

2501457440

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

have been identified as potentially carcinogenic has induced pulmonary cancer in animals under experimental conditions.[234]

Antismokers often refer to comparisons of sidestream and mainstream smoke constituents, or sidestream to mainstream smoke ratios, to support their claims about ETS. (A ratio here means the number obtained by dividing the amount of a constituent in sidestream smoke by the amount of that same constituent in mainstream smoke. For example, a ratio of 10 for nicotine would indicate that there is 10 times as much nicotine in sidestream as in mainstream smoke.) However, such a ratio is derived from laboratory experiments which usually employ smoking machines and experimental chambers to measure samples taken from the burning ends of cigarettes. Given these conditions, it is not surprising that some levels of sidestream smoke constituents may be greater than those in mainstream smoke. Moreover, as mentioned above, these experimental techniques do not take into account the impact of dilution which occurs under more realistic settings. Nor do they take into account the fact that even slight variations in the moisture content of tobacco or the porosity of the cigarette paper may greatly affect the reported ratios.[228]

Thus, it is both inappropriate and misleading to suggest that sidestream smoke is equivalent to ETS and that, under ordinary conditions, the nonsmoker is exposed to the levels of constituents reported in sidestream smoke.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Constituents of ETS

A number of constituents typically have been cited in the literature to demonstrate the contribution of ETS to the indoor air. These include carbon monoxide, particulates, nicotine, nitrosamines and others. However, the use of one or any combination of such constituents to determine ETS levels presents many problems. For example, although analytic and sampling methods continue to improve, there is at present no completely satisfactory and uniform procedure for measuring ETS.[235-236] Moreover, although the findings of studies which measure constituents in experimental conditions (e.g., in unventilated smog chambers) are cited to dramatize the potential effect of ETS on indoor air, they have little, if any, similarity to those studies which measure ETS in realistic settings. And finally, with the exception of nicotine, none of the constituents which have been used as substitute measures for ETS are characteristic of ETS alone. Other sources, such as heaters, stoves and furnishings, generate substances at levels greater than those found in ETS.

Carbon Monoxide

The scientific literature on carbon monoxide (CO) from environmental tobacco smoke contains numerous examples of studies conducted under unrealistic conditions. For instance, in one such

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

experiment, 80 cigarettes and 2 cigars were burned during a 78-minute period in a small unventilated simulated conference room.[237] In another case, 9 cigarettes were smoked one after another in an unventilated automobile.[238] Not surprisingly, the levels of CO measured in these unusual situations were extraordinarily high. Regrettably, studies of that sort are often cited as conclusive evidence of the impact of ETS on ordinary room air.

On the other hand, when carbon monoxide from ETS has been measured under realistic conditions, its contributions have been determined to be minimal.[239-246] Most recently, scientists in the United Kingdom monitored CO and other constituents in over 3,000 locations throughout the country.[246] They reported that carbon monoxide levels did not differ significantly between smoking and nonsmoking environments. Putting such studies into perspective, Dr. Duncan Hutcheon, a professor of medicine who has reviewed the literature, noted: "Environmental studies suggest that tobacco smoke has little impact on the CO content of the room air except under highly artificial conditions."[247]

What then are the sources of CO in public places? Research has shown that the main sources in the outdoor urban environment are motor vehicles and industrial processes,[248] and that indoor levels are affected by these outdoor sources, mainly through ventilation, and by numerous activities such as cooking and heating. In fact, studies have indicated that gas stoves in kitchens and heating units are often major sources of CO in homes.[249-251]

2501457443

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

After their review of such studies, participants at the Geneva symposium on ETS concluded that CO from environmental tobacco smoke "is not important from a health point of view."[16] Similarly, researchers from the Lawrence Berkeley Laboratory in the U.S., who are otherwise critical of tobacco smoke, concluded that "based on theoretical and empirical results, CO sidestream emissions from cigarettes have often been overemphasized."[252]

Particulates

A paper published in a 1980 issue of Science magazine, in which the authors reported the results of their efforts to measure particles or particulates in the air of smoking and nonsmoking areas, is often cited to support the claim that ETS is a major indoor pollutant.[253] The authors, Repace and Lowrey, contend that the levels of particles they observed in the smoking areas were much higher than in the nonsmoking areas. However, their study results are inconsistent with many others. For example, the average particle count attributed to ETS in their study was nearly twice as high as that determined in a study of 44 offices,[254] and nearly three to five times higher than the average levels reported in other studies of office buildings, restaurants and residences.[225,246,255-260]

There are a number of explanations for the authors' apparent overestimation of ETS exposure. First, they selectively

2501457444

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

sampled environments such as meeting and game rooms, bars and sandwich shops which did not represent normal occupancy conditions and where particulate levels would likely be high regardless of the presence or absence of tobacco smoke. Second, through inappropriate testing methods, they incorrectly assumed all particles in the air arose from ETS. However, as several researchers have noted, ETS typically contributes less than half to overall particle levels in indoor spaces.[261-263] Moreover, particles also are generated by people and their everyday routine activities such as movement and cooking.[264-265]

In addition to ignoring potential indoor sources of particles, the study has been criticized on other grounds. For example, Repace and Lowrey did not measure ventilation rates or humidity and they failed to calibrate properly their instrument prior to testing.[265] Indeed, the testing device they used "is no longer recommended" for tobacco smoke measurements.[235-236]

Repace and Lowrey implied that the particle levels attributed to ETS are potentially harmful to the health of nonsmokers. However, an earlier study by Bouhuys, et al., found "no evidence that high TSP (total suspended particulates) levels in homes with smokers were associated with increased symptoms or lung function loss among nonsmokers in the same home."[266] Binder, et al., found that respirable particulate levels in homes were "independent of the presence or absence of respiratory disease."[171] More recently, Lebowitz, et al., reported on data which showed

2501457445

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

that respiratory symptoms in asthmatics were associated with total particle levels indoors (e.g., dust), but not with the presence of tobacco smoke.[138,205]

Nicotine

Since environmental nicotine is produced exclusively by burning tobacco, it is considered to be a more reliable indicator of the amount of tobacco smoke in the environment than other smoke constituents. Studies which have used nicotine in this way suggest that tobacco smoke contributes little to the indoor atmosphere.[242,246,257-260,267-272]

For example, Drs. William Hinds and Melvin First of the Harvard School of Public Health found very small amounts of nicotine in the atmospheres of bars, bus and airline terminals, restaurants, and cocktail and student lounges.[269] In a 1980 publication, Dr. Hinds wrote that in public places, "the typical, average airborne concentration" of the tobacco smoke to which the nonsmoker is exposed is equivalent to a small fraction of a cigarette per hour.[273]

French researchers, using a different method of nicotine measurement to assess the amount of tobacco smoke in the atmosphere, reported higher concentrations of nicotine than Hinds and First.[267] However, they still concluded that "smoking does not present a risk to nonsmokers."

2501457446

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

In 1984, Japanese researchers tested a personal nicotine monitor in a number of public places, including offices, restaurants, lobbies, terminals and public transportation. They reported levels of nicotine exposure equivalent to one one-thousandth (1/1000) to four one-hundredths (4/100) of a cigarette per hour.[271] Their findings were repeated in 1987.[272]

In 1986, scientists from the R.J. Reynolds Tobacco Company developed a portable sampler for nicotine, particulates and CO. In conjunction with ACVA International, a firm specializing in the assessment of indoor air quality, samples of nicotine, particulates and CO were taken in 23 office buildings and 48 restaurants in New York City. The scientists reported such low levels that a typical New Yorker would have to work 550 uninterrupted hours, or dine continuously for 400 hours, in order to be exposed to the nicotine equivalent of one cigarette.[257] In 1987, scientists from IT Corporation, a firm specializing in the assessment and reduction of environmental substances, repeated the New York study in offices and restaurants in Dallas, Texas and in Ottawa, Canada.[258,259] They reported average nicotine exposure levels equivalent to three one-hundredths (3/100) of a cigarette per eight-hour workday, and three one-thousandths (3/1000) of a cigarette during a one-hour meal.[258]

In a recent nationwide sampling survey in the United Kingdom, researchers monitored nearly 3000 sites in travel, work, home and leisure locations for ambient nicotine, CO and particle

2501457447

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

levels.[246] Smoking was known to have occurred at almost half of those sites, yet in three-fourths of the samples, nicotine levels were too low to be detected. Canadian researchers also reported levels of nicotine at or below levels of detection even in locations with recirculated air from smoking designated areas.[260]

There is little to suggest that the small amount of nicotine to which a nonsmoker may be exposed is related to human disease. For example, two German scientists monitored several physiological responses in nonsmokers exposed to tobacco smoke under laboratory conditions.[274] They concluded that the amount of nicotine to which their subjects were exposed was too small to alter sensitive test measurements of heart rate, heart muscle tracings (EKG), blood pressure or skin temperature. Similar findings were reported in 1982 by a Japanese physiologist.[275] More recent research tends to support these conclusions. In a 1983 study of nonsmoking flight attendants, researchers measured exposures to nicotine during transpacific flights. The researchers concluded that the concentrations of nicotine achieved were so small that they were "unlikely to have physiologic effects."[276] In 1986, researchers monitored levels of nicotine, CO and particulates in 66 commercial flights in the U.S. They concluded that the non-smoking sections of commercial aircraft were essentially free of ETS.[277]

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Nitrosamines

Nitrosamines have been described as substances present everywhere in the environment, and, consequently, as detectable "wherever they have been searched for."[278] This includes the air we breathe, the food we eat and the water we drink. One scientist stated that nitrosamines occur everywhere "independent of any type of tobacco combustion, in which case concentrations are often reached in respiratory air that are far above those that may be produced by tobacco smoke."[279] Another scientist, Dr. Helmut Schievelbein, Director of the Institute of Clinical Chemistry in Munich, observed that concentrations of nitrosamines either consumed or generated daily by individuals are "many times greater" than those found in environmental tobacco smoke.[280]

The claim has been made, largely based on reports by Brunnemann, et al., that nitrosamines from tobacco smoke represent a cancer risk to nonsmokers.[281-282] It is important to note, however, that Brunnemann and his co-workers have emphasized that there are at present "no epidemiological data linking human respiratory cancers to volatile nitrosamines."[283] Even the 1979 U.S. Surgeon General's Report raised questions about the relevance of their findings.[146]

It is sometimes claimed that nitrosamine levels in sidestream smoke exceed those found in mainstream smoke, with the implication that those levels are also found in ETS. This, how-

2501457449

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

ever, is not the case. In 1982, Austrian scientists reported that measurements of nitrosamine levels in ordinary public places were far below the values reported earlier by Brunnemann and his co-workers. They emphasized that the levels of nitrosamines which may occur in environmental tobacco smoke "are quite different" from those found in sidestream smoke.[284]

A reviewer of the literature in this area noted that the "experimental basis for carcinogenic effect on human beings has not been established for the very low concentrations" of nitrosamines to which nonsmokers are exposed.[242] A German scientist who discussed this point concluded that, as a consequence, any claimed implications of health risks from nitrosamines in tobacco smoke "clearly move into the range of speculation."[279]

Other Constituents

Other constituents which have been identified in analyses of sidestream smoke are often assumed to contribute substantially to indoor air in the form of ETS. The most commonly mentioned constituents include nitrogen dioxide (NO_2), formaldehyde and volatile organic compounds. However, these compounds are present in very small amounts in ETS.[285-287] Recent studies report no correlations between ETS and levels of NO_2 and volatile organic compounds in residences and offices.[288,289] Indeed, research indicates that levels of these components generated from other

2501457450

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

ordinary sources (e.g., cooking stoves, heaters and building materials) are much greater than those contributed by cigarette smoking.[285,290]

Cotinine as a Marker for ETS Exposures

In 1984, Japanese researchers reported that cotinine, a substance converted from nicotine by the body, could be used to measure nonsmoker exposure to ETS.[291] While a number of other reports appear to endorse the claim that cotinine is a reliable marker for total exposures to ETS,[292-297] others do not.[298-304] For example, researchers have found that individuals metabolize nicotine in different ways at different times and that elimination rates for cotinine vary among individuals.[298,299,303,304] In addition, recent research indicates that diet may contribute to levels of nicotine and cotinine found in the body.[305]

Scientists have also noted that different methods of analysis may influence final recorded levels of cotinine.[301,303,306] The 1984 Japanese study was criticized because the researchers used a method of analysis which was not specific to cotinine and which subsequently yielded cotinine estimates ten times higher than those reported by other researchers for the same levels of exposure.[306,307]

Thus, scientific research casts serious doubt on the use of cotinine as a marker for total ETS exposure. A uniform

2501457451

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

method of analysis of cotinine must be developed and the extent of individual variability in metabolism must be determined before any pronouncements concerning the efficiency of cotinine as a marker for ETS can be made.

Summary

The foregoing literature survey demonstrates that, at most, tobacco smoke has only a minor influence upon ordinary indoor environments compared to common household or workplace sources, such as stoves, heaters and furnishings. Yet tobacco smoke, because it is so easily identifiable, has become a target for those who apparently want a quick, simple solution to the problem of indoor air pollution. However, as one Canadian scientist observed, "the claim that smoking is responsible for indoor air pollution is an oversimplification of a complex, multi-source problem."[265]

2501457452

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

ENVIRONMENTAL TOBACCO SMOKE AND THE PUBLIC

Exposure to environmental tobacco smoke under extreme conditions may provoke complaints of irritation and annoyance. However, the mere sight and smell of tobacco smoke may be enough to produce strong reactions in individuals who intensely dislike smoking. Although the extent to which environmental tobacco smoke may bother or annoy individuals under normal conditions has not been established scientifically[16], it has been suggested that tobacco smoke is at most a minor annoyance for many individuals.[308-310] It has also been suggested that extreme reactions to tobacco smoke exposure may have a strong emotional or psychological basis.[311-312] Whatever the reasons for such reactions, the literature on this subject suggests it is misleading to claim, as antismokers frequently do, that such subjective or emotional reactions have a physical basis.

Governmental Regulations and Individual Rights

Although antismoking organizations claim to seek protection for the health and welfare of the nonsmoker, their efforts to make smoking socially unacceptable are part of a broader objective -- the severe restriction or even prohibition of smoking by governmental legislation. Of course, such regulations restrict the freedom of the smoker, and antismokers are compelled to justify this kind of political control of individual behavior. They have

2501457453

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

accomplished this, in large measure, by exploitation of the so-called "health" argument involving ETS exposures to nonsmokers.

For example, the most recent U.S. Surgeon General's Report on this subject contends that ETS causes lung cancer in adult nonsmokers and respiratory conditions in children, and that separation of smokers and nonsmokers is not effective in minimizing the nonsmoker's exposure to ETS[14]. The Report concludes that smoking bans will not only reduce ETS exposures, but will "alter smoking behavior and public attitudes about tobacco use." The Report further suggests that "over time, this may contribute to a reduction of smoking." Thus, the underlying motivation for the use of the ETS/health argument is to attain a "smoke-free society by the year 2000."

The Surgeon General's tactics, however, have been questioned by a number of critics. One reviewer suggested that the Surgeon General's conclusions were based on "flimsy" evidence presented in an effort to "divert attention" from important health concerns such as the "poisoning of the environment."[313] A U.S. Congressman, in a letter to the Congressional Record, wrote that "the conclusions in the Surgeon General's Report are not supported by the research in his own report."[314] Moreover, recent scientific studies aboard commercial aircraft and in offices indicate, contrary to the Surgeon General's Report, that separation of smokers and nonsmokers effectively minimizes ETS exposures for nonsmokers.[260,277]

2501457454

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Five U.S. labor union presidents criticized the Surgeon General's Report for minimizing the risk imposed by workplace toxins, for shifting the burden of cleaning up the workplace to workers, and for dismissing the importance of engineering controls (e.g., ventilation) in the maintenance of a safe and comfortable workplace.[315] The U.S. Tobacco Institute, in a comprehensive review of the issue, observed that premature scientific conclusions about ETS, together with increasing political pressure to accept the Surgeon General's conclusions, have "brought scientific integrity to a crossroads."[316]

However, some governmental bodies have refused to give in to the emotional rhetoric of antismoking organizations and public health officials. For example, courts in the United States have, with few exceptions, denied the claims of individuals who contend they have a "right" to a smoke-free workplace. A law journal article described this trend as representing "substantial judicial reluctance to supplant the roles of employer and employee in addressing smoking in the private workplace."[317]

In 1987, the Supreme Court of Canada refused an appeal by a worker who sought a smoke-free environment on the grounds that ETS is a "dangerous" workplace substance.[318] Similarly, in Japan, a court judgment denied a plaintiff's request that more than half of all the railway cars on passenger trains be designated nonsmoking because, the Court ruled, ETS exposures do not "exceed the limits of normal tolerance."[319]

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

In 1984, the U.S. Civil Aeronautics Board (CAB), the federal agency responsible at that time for regulating smoking aboard aircraft, considered a petition calling for a ban on smoking on flights lasting two hours or less. The CAB assessed the health claims advanced by many antismoking organizations, but denied the petition, arguing that there was little scientific basis to the claims.[320]

In 1987, the U.S. Department of Transportation rejected a recommendation from the National Academy of Sciences to prohibit smoking aboard commercial airliners[321] because it was not supported by data associating health effects and ETS exposures aboard aircraft.[322] Nonetheless, the U.S. Congress voted to ban smoking aboard domestic flights of two hours or less until 1990 when the law expires. The Department of Transportation subsequently issued a request for proposals for in-flight monitoring of ETS and other indoor air constituents.[323] The results will be presented to Congress for its deliberations in 1990. In the meantime, other research on this issue continues. In 1988, for example, a review of the relevant data concluded that "the available scientific evidence does not support the prohibition of smoking on commercial aircraft." It also stated that the available data "suggest that factors or substances other than ETS may be major contribution to subjective complaints of discomfort by passengers and flight crew."[324]

2501457456

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Other governmental agencies, on the other hand, have done little to clarify the issue for the public. In the United States, for instance, two separate government-sponsored reports recommended restricting tobacco smoking on the basis of risks that were characterized as "biologically plausible".[93,321] Another report sought to identify the known facts and areas of uncertainty regarding the ETS issue and observed that "many more of the latter were found than the former." The report nevertheless concluded that exposures to ETS harm the health of nonsmokers.[13] Similarly, the most recent monograph on tobacco smoke from the WHO's International Agency for Research on Cancer (IARC) warns of possible health consequences from exposures to ETS while conceding that the scientific evidence is consistent "either with an increase or with an absence of risk."[325]

In the United Kingdom, the Fourth Report of the Independent Scientific Committee on Smoking and Health published in 1988 endorsed the heavily criticized claim that nonsmoker exposure to ETS increases the risk of lung cancer from 10 to 30 percent compared with nonsmokers who are not exposed[15], despite the fact that, as one commentator noted, "exposure data are very unreliable, and there is a proven basis for bias in the direction of exaggeration of the risk."[326]

In 1985, the Supreme Insurance Court of Sweden awarded compensation for occupational injury to the estate of a nonsmoker who worked with smokers and who subsequently died of lung cancer.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Although the media described the Court's decision as a causal indictment of ETS exposure, one commentator noted that the decision was based upon the structure of occupational injury regulations in Sweden and not upon any scientific evidence of a causal relationship between lung cancer and ETS exposures.[327] This author explained that the Swedish insurance system operates under a reverse burden-of-proof provision, namely, that the relationship between a suspected factor and an injury is assumed to be causal unless substantially stronger reasons are produced to suggest otherwise. Consequently, this provision does not require the claimant to present scientific proof of a causal relationship between a suspected factor and an injury.

Three years later, in Australia, a city bus driver who contended his lung cancer was caused, at least in part, by exposure to ETS on the job was awarded a claim by the insurer of the Melbourne Transit Authority.[328] This settlement was interpreted by some as establishing legal precedent for the claim that ETS exposure has been proven to cause lung cancer. However, the bus driver's claim regarding ETS was only part of his complaint and the tribunal before which the proceeding was held heard only part of the evidence in the case before the insurer made a settlement offer.[329] Thus, this settlement established no formal legal precedent regarding ETS health claims.[328,329]

2501457458

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

An Australian writer recently summarized the issue of governmental influence on the ETS question.[330] He wrote:

> Those involved in government anti-smoking activities should be aware of the tenuous nature of the data on passive smoking and health effects. The deliberate use of fear inducing tactics by misrepresenting such data by dubious extrapolations is propaganda and not health education.

It is clear that once the health claims regarding environmental tobacco smoke are placed in their proper scientific perspective, the issue of prohibiting smoking in public places becomes a social and political one. As a former director of the WHO wrote recently, "the question as to whether the conceivable theoretical possibility of a risk calls for official preventive measures is not a medical but a politico-social one."[19]

The issue thus involves whether or not public policy should mandate prohibition of smoking because it is seen by some as a nuisance or annoyance. If the decision is reached in favor of regulation, then one must decide whether the same line of reasoning applies to other kinds of "annoyances" encountered in everyday life and whether those "annoyances" should also be banned. The implication of such reasoning should be obvious to anyone who is opposed to unwarranted governmental intrusion into people's lives.

2501457459

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

What is the Solution?

A recent article on the principles of public policy relating to smoking suggests an alternative to governmental regulations. According to this article, "the appropriate form of public policy is the promotion of courtesy and cooperation between smokers and nonsmokers, rather than outright prohibition."[331] This solution relies upon common sense, courtesy and the free marketplace for decisions concerning smoking. More importantly, however, this solution avoids unnecessary governmental interference with its subsequent loss of individual freedom.

2501457460

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

REFERENCES*

1. Shopland, D., Quoted in "The Campaign Against Smoking Gains Momentum," New Engl J Med 314(16): 1059-1064, 1986.

2. Berger, P., "A Sociological View of the Antismoking Phenomenon," Smoking and Society, ed. R. Tollison (Lexington: D.C. Heath and Co., 1986): 225-240.

3. Warner, K., "Toward a Global Strategy to Combat Smoking: The 5th World Conference on Smoking and Health," J Public Health Policy 5(1): 28-39, 1984.

4. Hulka, B., Quoted in "Other People's Smoke May Be Risky for You," NRC News Report 37(1): 8-11, 1986-1987.

5. Witorsch, P., "Passive Smoking," New Zealand Med J: 258-9, April 22, 1987.

6. Amiel, B., "Smoke in Their Eyes," The (London) Times: 15, March 13, 1987.

7. Kris, M., "High Risk of Cancer for Passive Smokers. More Than Much Ado About Nothing?," Aertze Zeiting 5(220): 1-2, December 11, 1986.

8. Adlkofer, Franz, "The Problems of Passive Smoking," Der Kassenarzt 27(51/52): 29-39, 1987. Translation.

9. Uberla, K., "Lung Cancer from Passive Smoking: Hypothesis or Convincing Evidence?," Int Arch Occup Environ Health 59: 421-437, 1987.

10. Hirayama, T., "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer: A Study from Japan," Br Med J I, 282: 183-185, 1981.

11. Trichopoulos, D., et al., "Lung Cancer and Passive Smoking," Int J Cancer 27(1): 1-4, 1981.

12. White, J. and H. Froeb, "Small-Airways Dysfunction in Nonsmokers Chronically Exposed to Tobacco Smoke," New Engl J Med 302(13): 720-723, 1980.

---

*All translations used in preparing this document are "unofficial" and may be subject to modification.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

13. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects (Washington: National Academy Press, 1986).

14. U.S. Department of Health and Human Services, The Health Consequences of Involuntary Smoking: A Report of the Surgeon General (U.S. DHHS: Washington, 1986).

15. Department of Health and Social Security, Fourth Report of the Independent Scientific Committee on Smoking and Health (London: Her Majesty's Stationery Office, 1988).

16. Rylander, R., "Workshop Perspectives," ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels, eds. R. Rylander, et al., Eur J Respir Dis, Suppl. 133(65): 143-145, 1984.

17. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, "Report of Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies, May 1-3, 1983," December, 1983.

18. Valentin, H. and E. Wynder, "Health Danger Through Passive Smoking Not Proven: Physicians View on Passive Smoking," (Press release for Symposium on "Passive Smoking from a Medical Point of View," April 9-12, 1984, Vienna, Austria).

19. Handler, J., Preface, Passivrauchen aus medizinischer Sicht [Medical Perspectives on Passive Smoking] (Geneva: IRL Imprimeries Reunies Lausanne, 1986): A-B.

20. "Proceedings of the International Experimental Toxicology Symposium on Passive Smoking," ed. E. Mohtashamipur E., Toxicology Letters 35(1): 1-168, 1987.

21. Gostomzyk, G.J., "Passive Smoking-Report on an International Symposium (23-25 October 1986)," Public Health 49: 212-215, 1987.

22. Kasuga, Hitoshi, et al., "International Conference on Indoor Air Quality, Tokyo, 1987," Nikkei Medical 1(10): 168-175, 1988. Translation.

23. Lehnert, G. and E. Wynder, "Illness Due to Passive Smoking?," (Press Release, Vienna, Austria: May 3, 1988). Translation.

24. Witorsch, P., "Passive Smoking," New Zealand Med J: 865, November 12, 1986.

2501457462

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

25. Aviado, D., "Health Issues Relating to 'Passive Smoking'," Smoking and Society, Chapter 4: 158, 1985.

26. Lee, P., et al., "Relationship of Passive Smoking to Risk of Lung Cancer and Other Smoking-Associated Diseases," Brit J Cancer 54: 97-105, 1986.

27. Aviado, D.M., "Toxicological Basis for Regulation of Indoor Air Quality," J Applied Toxicol 8(3): 155-157, 1988.

28. Ahlborn, W. and K. Uberla, "Passive Smoking and Lung Cancer: Reanalyses of Hirayama's Data," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 169-178.

29. Diamond, G. and J. Forrester, "Clinical Trials and Statistical Verdicts: Probable Grounds for Appeal," Ann Intern Med 98(3): 385-394, 1983.

30. Gostomzyk, J., "Krank durch Passivrauchen? (Disease Due to Passive Smoking?)," Munch med Wschr 124(4): 11, 1982. Translation.

31. Grimmer, G., "Kausalitätsbeziehung Fraglich (Causality-Relationship Questionable)," Munch med Wschr 124(4): 16, 1982. Translation.

32. Grundmann, E., et al., "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer," Br Med J I 282: 1156, 1981.

33. Kilpatrick, S.J. and J. Viren, "Age as a Modifying Factor in the Association Between Lung Cancer in Non-smoking Women and Their Husbands' Smoking Status," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 195-202.

34. Lee, P., "Passive Smoking," Food Chem Toxicol 20: 223-229, 1982.

35. Lehnert, G., "Krank durch Passivrauchen? (Disease Due to Passive Smoking?)," Munch med Wschr 123(40): 1485-1488, 1981. Translation.

36. MacDonald, E., "Non-Smoking Wives of Heavy Smokers Have a High Risk of Lung Cancer," Br Med J II 283: 915-916, 1981.

2501457463

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

37. MacDonald, E., "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer," Br Med J II 283: 1465, 1981.

38. Mantel, N., "Epidemiologic Investigations: Care in Conduct, Care in Analysis, and Care in Reporting," J Cancer Res Clin Oncol 105: 113-116, 1983.

39. Rutsch, M., "Mangelhafte Statistische Absicherung (Inadequate Statistical Substantiation)," Munch med Wschr 124(4): 15, 1982. Translation.

40. Rutsch, M., "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer," Br Med J I 282: 985, 1981.

41. Schievelbein, H., "Kein Beweis für Gesundheitsschädlichkeit (No Proof of Injuriousness to Health)," Munch med Wschr 124(4): 13-15, 1982. Translation.

42. Schmähl, D., Quoted in "Lungenkrebs durch Passivrauchen? Studie aus Japan bestätigt Verdacht/Widerspruch aus Deutschland (Lung Cancer from Passive Smoking? Study from Japan Confirms Suspicion/Contradictory Opinion from Germany)," Süddeutsch Zeitung, March 18, 1981. Translation.

43. Sterling, T., "Non-Smoking Wives of Heavy Smokers Have a High Risk of Lung Cancer," Br Med J I 282: 1156, 1981.

44. Rylander, R., "Environmental Tobacco Smoke and Lung Cancer," ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels, eds. R. Rylander, et al., Eur J Respir Dis, Suppl.133(65): 127-133, 1984.

45. Trichopoulos, D., et al., "Lung Cancer and Passive Smoking: Conclusions of Greek Study," Lancet II: 677-678, 1983.

46. Garfinkel, L., "Time Trends in Lung Cancer Mortality Among Nonsmokers and a Note on Passive Smoking," JNCI 66(6): 1061-1066, 1981.

47. Lam, T.H., et al., "Smoking, Passive Smoking and Histological Types in Lung Cancer in Hong Kong Chinese Women," Br J Cancer, 56(5): 673-678, 1987.

48. Pershagen, G., et al., "Passive Smoking and Lung Cancer in Swedish Women," Am J Epidemiol 125(1): 17-24, 1987.

49. Akiba, S., et al., "Passive Smoking and Lung Cancer Among Japanese Women," Cancer Res 46: 4804-4807, 1986.

2501457464

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

50. Humble, C.G., et al., "Marriage to a Smoker and Lung Cancer Risk," AJPH 77(5): 598-602, 1987.

51. Brownson, R.C., et al., "Risk Factors for Adenocarcinoma of the Lung," Am J Epidemiol 125(1): 25-34, 1987.

52. Knoth, A., et al., "Passive Smoking as a Causative Factor of Lung Cancer in Nonsmoking Women," Med Klin Prax 78(2): 56-59, 1983. Translation.

53. Heller, W., "Lung Cancer and Passive Smoking," Lancet II: 1309, 1983.

54. Correa, P., et al., "Passive Smoking and Lung Cancer," Lancet II: 595-597, 1983.

55. Buffler, P., et al., "The Causes of Lung Cancer in Texas," Lung Cancer: Causes and Prevention, eds. Mizell and Correa (Deerfield Beach: Verlag Chemie International, 1984): 83-99.

56. Repace, J. and A. Lowrey, "A Quantitative Estimate of Nonsmokers' Lung Cancer Risk from Passive Smoking," Environ Int 11(1): 3-22, 1985.

57. Marshall, E., "Tobacco Science Wars," Science 236: 250-251, 1987.

58. Arundel, A., et al., "Nonsmoker Lung Cancer Risks from Tobacco Smoke Exposure: An Evaluation of Repace and Lowrey's Phenomenological Model," J Environ Sci Health C4(1): 93-118, 1986.

59. Anonymous, "The Murky Hazards of Secondhand Smoke," Consumer Reports: 81-84, February, 1985.

60. Arundel, A., et al., "Never Smoker Lung Cancer Risks from Exposure to Particulate Tobacco Smoke," Environ Int 13: 409-426, 1987.

61. Lebowitz, M., "The Potential Association of Lung Cancer with Passive Smoking," Environ Int 12: 3-9, 1986.

62. Johnson, C., "Letter to the Editors," Environ Int 12: 21-22, 1986.

63. Burch, P., "Health Risks of Passive Smoking: Problems of Interpretation," Environ Int 12: 23-28, 1986.

2501457465

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

64. Kilpatrick, S.J., Jr., "Letter to the Editors," Environ Int 12: 29-31, 1986.

65. Miller, G., "Cancer, Passive Smoking and Nonemployed and Employed Wives, West J Med 140(4): 632-635, 1984.

66. Arundel, A. and T. Sterling, "Review of the Epidemiological Studies on Lung Cancer and Nonsmoker's Exposure to Environmental Tobacco Smoke," Private Communication to the Special Projects Office, U.S. OTA Health Program, April, 1986.

67. Sandler, D., et al., "Cancer Risk in Adulthood from Early Life Exposure to Parents' Smoking," Am J Public Health 75(5): 487-492, 1985.

68. Sandler, D., et al., "Cumulative Effects of Lifetime Passive Smoking on Cancer Risk," Lancet II: 312-314, 1985.

69. Sandler, D., et al., "Passive Smoking in Adulthood and Cancer Risk," Am J Epidemiol 121(1): 37-48, 1985.

70. Lee, P., "Lifetime Passive Smoking and Cancer Risk," Lancet II: 1444, 1985.

71. Burch, P., "Lifetime Passive Smoking and Cancer Risk," Lancet II: 866, 1985.

72. Burch, P., "Passive Smoking in Adulthood and Cancer Risk," Am J Epidemiol 123(2): 368-9, 1986.

73. Higgins, I., "Lifetime Passive Smoking and Cancer Risk," Lancet II: 866-867, 1985.

74. Friedman, G., "Passive Smoking in Adulthood and Cancer Risk," Am J Epidemiol 123(2): 367, 1986.

75. Mantel, N., "Passive Smoking in Adulthood and Cancer Risk," Am J Epidemiol 123(2): 367-8, 1986.

76. Vandenbroucke, J., et al., "Active and Passive Smoking in Married Couples: Results of 25 Year Follow Up," Br Med J I: 1801-1802, 1984.

77. Gillis, C., et al., "The Effect of Environmental Tobacco Smoke in Two Urban Communities in the West of Scotland," ETS-Environmental Tobacco Smoke. Report from a Workshop on Effects and Exposure Levels, eds. R. Rylander, et al., Eur J Respir Dis, Suppl. 133(65): 121-126, 1984.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

78. Kabat, G. and E. Wynder, "Lung Cancer in Nonsmokers," Cancer 53(5): 1214-1221, 1984.

79. Garfinkel, L., et al., "Involuntary Smoking and Lung Cancer: A Case-Control Study," JNCI 75(3): 463-469, 1985.

80. Chan, W. and S. Fung, "Lung Cancer in Non-Smokers in Hong Kong," Cancer Campaign Vol. 6, Cancer Epidemiology, ed. E. Grundmann (Stuttgart: Gustav Fischer Verlag, 1982): 199-202.

81. Koo, L., et al., "Is Passive Smoking an Added Risk Factor for Lung Cancer in Chinese Women?," J Exp Clin Cancer Res 3(3): 277-283, 1984.

82. Koo, L., et al., "Measurements of Passive Smoking and Estimates of Lung Cancer Risk Among Non-Smoking Chinese Females," Int J Cancer 39: 162-169, 1987.

83. Koo, L., et al., "Life-History Correlates of Environmental Tobacco Smoke: A Study on Nonsmoking Hong Kong Chinese Wives with Smoking Versus Nonsmoking Husbands," Soc Sci Med 26(7): 751-760, 1988.

84. Koo, L., "Dietary Habits and Lung Cancer Risk Among Chinese Females in Hong Kong Who Never Smoked," Nutr Cancer 11: 155-172, 1988.

85. Wu, A., et al., "Smoking and Other Risk Factors for Lung Cancer in Women," JNCI 74(4): 747-751, 1985.

86. Lloyd, O., et al., "Respiratory Cancer in a Scottish Industrial Community: A Retrospective Case-Control Study," J Soc Occup Med 36: 2-8, 1986.

87. Wald, N.J., et al., "Does Breathing Other People's Tobacco Smoke Cause Lung Cancer?," Br Med J 293: 1217-1222, 1986.

88. Blot, W. and J. Fraumeni, Jr., "Passive Smoking and Lung Cancer," JNCI 77: 993-100, 1986.

89. Doll, R., "Lung Cancer: Observed and Expected Changes in Incidence from Active and Passive Smoking," Presentation to UICC Conference, Budapest, August, 1986.

90. Katzenstein, A., Environmental Tobacco Smoke (ETS) and Risk of Lung Cancer--How Convincing is the Evidence?, (Katzenstein Associates, Larchmont, NY, March, 1987.)

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

91. Mantel, N., "Passive Smoking and Disease," Statistics Colloquium, The American University, May 4, 1988.

92. Letzel, H., et al., "Meta-Analyses on Passive Smoking and Lung Cancer Effects on Study Selection and Misclassification of Exposue," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 293-302.

93. Staff Paper, Special Projects Office of the Health Program, OTA, "Passive Smoking in the Workplace: Selected Issues," Office of Technology Assessment, United States Congress, May, 1986.

94. Schwartz, S.L. and N.J. Balter, "ETS-Lung Cancer Epidemiology: Supportability of Misclassification and Risk Assumptions," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 159-168.

95. Lee, P., "Passive Smoking and Lung Cancer: Problems in Interpreting the Epidemiological Data," Presentation to Toxicology Forum, Washington, D.C., February, 1987.

96. Lee, P., "Does Breathing Other People's Tobacco Smoke Cause Lung Cancer?," Br Med J 293: 1503-1504, 1986.

97. Lee, P., "Misclassification as a Factor in Passive Smoking Risk," Lancet II: 867, 1986.

98. Lee, P., "Lung Cancer and Passive Smoking: Association an Artefact Due to Misclassification of Smoking Habits?," Toxicology Letters 35: 157-162, 1987.

99. Lee, P., "Passive Smoking and Lung Cancer Association: A Result of Bias?," Human Toxicol 6: 517-524, 1987.

100. Lee, P., "An Alternative Explanation for the Increased Risk of Lung Cancer in Non-Smokers Married to Smokers," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 149-158.

101. Kilpatrick, S.J., Jr., "Misclassification of Environmental Tobacco Smoke Exposure: Its Potential Influence on Studies of Environmental Tobacco Smoke and Lung Cancer," Toxicology Letters 35: 163-168, 1987.

102. Friedman, G., et al., "Prevalence and Correlates of Passive Smoking," Am J Public Health 73(4): 401, 1983.

103. Pron, G.E., et al., "The Reliability of Passive Smoking

2501457468

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

Histories Reported in a Case-Control Study of Lung Cancer," Am J Epidemiol 127(2): 267-273, 1988.

104. Schenker, M., et al., "Assessment of Environmental Tobacco Smoke Exposure in Epidemiologic Studies," Chest 91(2): 313-314, 1987. Abstract.

105. Lerchen, L. and J.M. Samet, "An Assessment of the Validity of Questionnaire Responses Provided by a Surviving Spouse," Am J Epidemiol 123(3): 481-489, 1986.

106. Sandler, D.P. and D.L. Shore, "Quality of Data on Parents' Smoking and Drinking Provided by Adult Offspring," Am J Epidemiol 124(5): 768-778, 1986.

107. Wynder, E., "Guidelines to the Epidemiology of Weak Associations," Prev Med 16: 211-212, 1987.

108. Mantel, N., "Lung Cancer and Passive Smoking," Br Med J 294: 440, 1987.

109. Lee, P., "Deaths in Canada from Lung Cancer Due to Involuntary Smoking," CMAJ 137: 372-373, 1987.

110. Gordis, Leon, "Challenges to Epidemiology in the Next Decade," Am J Epidemiol 128(1): 1-9, 1988.

111. Newcombe, Robert G., "Towards a Reduction in Publication Bias," Br Med J 295: 656-659, 1987.

112. DeSerres, F. J. and T. Matsushima, "Meeting Report: Mutagenesis and Carcinogenesis by Nitropyrenes and Cancer Chemotherapeutics," Mutat Res 164: 4, 1986.

113. Anonymous, "Lung Cancer in South China," Oncology Times, VIII (6): 33, 1986.

114. Mumford, J.N., et al., "Lung Cancer and Indoor Air Pollution in Xuan Wei, China," Science 235: 217-220, 1987.

115. Gao, Y.T., et al., "Lung Cancer Among Chinese Women," Int J Cancer 40: 604-609, 1987.

116. Kauffmann, F., et al., "Adult Passive Smoking in the Home Environment: A Risk Factor for Chronic Airflow Limitation," Am J Epidemiol 117(3): 269-280, 1983.

117. Adlkofer, F., et al., "Small-Airways Dysfunction in Passive Smokers," New Engl J Med 303(7): 392, 1980.

2501457469

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

118. Huber, G., "Small Airways Dysfunction in Passive Smokers," New Engl J Med 303(7): 392, 1980.

119. Freedman, A., "Small-Airways Dysfunction in Passive Smokers," New Engl J Med 303(7): 393, 1980.

120. Aviado, D., "Small-Airways Dysfunction in Passive Smokers," New Engl J Med 303(7): 393, 1980.

121. White, J. and H. Froeb, "Small-Airways Dysfunction in Passive Smokers," New Engl J Med 303(7): 393-394, 1980.

122. Lebowitz, M., "Forum: Second Hand Smoke -- How Dangerous?," Presentation to American Lung Association/American Thoracic Society Annual Meeting, Los Angeles, California, May 17, 1982.

123. Fountain, L.H., "White-Froeb Study Discredited by Scientists," U.S. Congressional Record, 97th Cong., 2nd Sess., December 16, 1982 (Washington: Government Printing Office, 1982): E5252-5254.

124. Lebowitz, M., "Influence of Passive Smoking on Pulmonary Function: A Survey," Prev Med 13(6): 645-655, 1984.

125. Kentner, M., et al., "The Influence of Passive Smoking on Pulmonary Function - A Study of 1,351 Office Workers," Prev Med 13(6): 656-669, 1984.

126. Kentner, M. and D. Weltle, "Passive Tobacco Smoke Inhalation and Lung Function in Adults," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 232-241.

127. Shephard, R., et al., "Responses of Exercising Subjects to Acute 'Passive' Cigarette Smoke Exposure," Environ Res 19(1): 279-291, 1979.

128. Comstock, G., et al., "Respiratory Effects of Household Exposures to Tobacco Smoke and Gas Cooking," Am Rev Respir Dis 124(2): 143-148, 1981.

129. Jones, J., et al., "Effects of Cooking Fuels on Lung Function in Nonsmoking Women," Arch Environ Health 38(4): 219-222, 1983.

130. Lebowitz, M., et al., "Family Aggregation of Pulmonary Function Measurements," Am Rev Respir Dis 129: 8-11, 1984.

2501457470

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

131. Schilling, R., et al., "Lung Function, Respiratory Disease, and Smoking in Families," Am J Epidemiol 106(4): 274-283, 1977.

132. Hosein, H. and P. Corey, "Domestic Air Pollution and Respiratory Function in a Group of Housewives," Can J Public Health 77: 44-50, 1986.

133. Dahms, T., et al., "Passive Smoking -- Effects on Bronchial Asthma," Chest 80(5): 530-534, 1981.

134. Pimm, P., et al., "Physiological Effects of Acute Passive Exposure to Cigarette Smoke in Asthmatics," Fed Proc 36(3): 606, 1977. Abstract.

135. Shephard, R., et al., "'Passive' Exposure of Asthmatic Subjects to Cigarette Smoke," Environ Res 20: 392-402, 1979.

136. Knight, A., and A. Breslin, "Passive Cigarette Smoking and Patients With Asthma," Med J Aust 142(3): 194-5, 1985.

137. Ing, A. and A. Breslin, "The Effect of Passive Cigarette Smoking on Asthmatic Patients," Proceedings, The Thoracic Society of Australia, October, 1983: 543. Abstract.

138. Lebowitz, M., "The Effects of Environmental Tobacco Smoke Exposure and Gas Stoves on Daily Peak Flow Rates in Asthmatic and Non-Asthmatic Families," ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels, Eur J Respir Dis, eds. R. Rylander, et al., Suppl. 133(65): 90-97, 1984.

139. Weidemann, H., et al., "Acute Effects of Passive Smoking on Lung Function and Airway Reactivity in Asthmatic Subjects," Chest 89(2): 180-185, 1986.

140. Hirayama, T., "Lung Cancer in Japan: Effects of Nutrition and Passive Smoking," Lung Cancer: Causes and Prevention, eds. Mizell and Correa (Deerfield Beach: Verlag Chemie International, 1984): 175-195.

141. Helsing, K.J., et al., "Heart Disease Mortality in Nonsmokers Living with Smokers," Am J Epidemiol 127(5): 915-922, 1988.

142. Svendsen, K.H., et al., "Effects of Passive Smoking in the Multiple Risk Factor Intervention Trial," Am J Epidemiol 126(5): 783-795, 1987.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

143. Garland, C., et al., "Effects of Passive Smoking on Ischemic Heart Disease Mortality of Nonsmokers," Am J Epidemiol 121(5): 645-50, 1985.

144. Schievelbein, H. and F. Richter, "The Influence of Passive Smoking on the Cardiovascular System," Prev Med 13(6): 626-644, 1984.

145. Aronow, W., "Effects of Passive Smoking on Angina Pectoris," New Engl J Med 299(1): 21-24, 1978.

146. U.S. Public Health Service, "Chapter 11, Involuntary Smoking," Smoking and Health: A Report of the Surgeon General, Department of Health, Education and Welfare, Washington, DHEW Publication No. 79-50066: 1-41, 1979.

147. Fisher, E., Statement, U.S., Congress, House, Committee on Agriculture, Subcommittee on Tobacco, Effect of Smoking on Nonsmokers, Hearing, 95th Cong., 2nd Sess., September 7, 1978 (Washington: Government Printing Office, 1978): 35-40.

148. Shephard, R., The Risks of Passive Smoking (London: Croom-Helm Ltd., 1982): 73.

149. McNicol, M., and J. A. McM Turner, "Oxygen Uptake at the Onset of Angina Pectoris: Effects of Nicotine and Carbon Monoxide," Clin Sci 65(3): 24, 1983. Abstract.

150. Mintz, M., "FDA, Citing Phony Evidence, Bars Drug Tests by Researcher," The Washington Post, March 23, 1983.

151. Peterson, C., "EPA Probe Criticizes a Study Used in Air Quality Standard," The Washington Post, June 7, 1983.

152. Camacho, E., et al., "Pulmonary Symptoms and Pulmonary Functional Tests Among Children in Relation to the Area of Residence," Eur Respir Dis 63(2): 165-166, 1982.

153. Lebowitz, M., et al., "The Effect of Passive Smoking on Pulmonary Function in Children," Environ Intl 8: 371-373, 1982.

154. Bland, M., et al., "Effect of Children's and Parents' Smoking on Respiratory Symptoms," Arch Dis Child 53(2): 100-105, 1978.

155. Bonham, G. and R. Wilson, "Children's Health in Families with Cigarette Smokers," Am Public Health 71(3): 290-293, 1981.

2501457472

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

156. Burchfiel, C., et al., "Passive Smoking in Childhood: Respiratory Conditions and Pulmonary Function in Tecumseh, Michigan," Am Rev Respir Dis 133(6): 966-73, 1986.

157. Cameron, P. and D. Robertson, "Effect of Home Environment Tobacco Smoke on Family Health," J Appl Psychol 57(2): 142-147, 1973.

158. Colley, J., "Respiratory Symptoms in Children and Parental Smoking and Phlegm Production," Br Med J II: 201-204, 1974.

159. Colley, J., et al., "Influence of Passive Smoking and Parental Phlegm on Pneumonia and Bronchitis in Early Childhood," Lancet II: 1031-1034, 1974.

160. Ekwo, E., et al., "Relationship of Parental Smoking and Gas Cooking to Respiratory Disease in Children," Chest 84(6): 662-668, 1983.

161. Fergusson, D., et al., "Parental Smoking and Lower Respiratory Illness in the First Three Years of Life," J Epidemiol Community Health 35(3): 180-184, 1981.

162. Fergusson, D., et al., "Parental Smoking and Respiratory Illness During Early Childhood," Ped Pul 1(2): 99-106, 1985.

163. Ferris, B., et al., "The Six-City Study: Examples of Problems in Analysis of Data," Environ Health Perspect 52: 115-123, 1983.

164. Gortmaker, S., et al., "Parental Smoking and the Risk of Childhood Asthma," Am J Public Health 72(6): 574-579, 1982.

165. Harlap, S. and A. Davies, "Infant Admissions to Hospital and Maternal Smoking," Lancet I: 529-532, 1974.

166. Kraemer, M., et al., "Risk Factors for Persistent Middle Ear Effusions: Otitis Media, Catarrh, Cigarette Smoke Exposure, and Atopy," JAMA 249(8): 1022-1025, 1983.

167. Ogston, S.A., et al., "Association of Infant Alimentary and Respiratory Illness with Parental Smoking and Other Environmental Factors," J Epidemiol Commun Health 41(1): 21-25, 1987.

168. Schenker, M., et al., "Risk Factors for Childhood Respiratory Disease: The Effect of Host Factors and Home Environ-

2501457473

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

mental Exposures," Am Rev Respir Dis 128: 1038-1043, 1983.

169. Tsimoyianis, G.V., et al., "Reduction in Pulmonary Function and Increased Frequency of Cough Associated With Passive Smoking in Teenage Athletes," Pediatrics 80(1): 32-36, 1987.

170. Ware, J., et al., "Passive Smoking, Gas Cooking, and Respiratory Health of Children Living in Six Cities," Am Rev Respir Dis 129: 366-374, 1984.

171. Binder, R., et al., "Importance of the Indoor Environment in Air Pollution Exposure," Arch Environ Health 31(6): 277279, 1976.

172. Bouhuys, A., "Lung Diseases in Rural and Urban Communities," Report Sponsored by the Division of Lung Diseases, National Heart, Lung and Blood Institute, U.S. National Institutes of Health, pp. 77-89, June 10, 1977.

173. Gardner, G., et al., "Effects of Social and Family Factors on Viral Respiratory Infection and Illness in the First Year of Life," J Epidemiol Community Health 38: 42-48, 1984.

174. Harrington, W. and A. J. Krupnick, "Short-Term Nitrogen Dioxide Exposure and Acute Respiratory Disease in Children," J Air Pollut Control Assoc 35: 1061-1067, 1985.

175. Holma, B. and O. Winding, "Housing, Hygiene, and Health: A Study in Old Residential Areas in Copenhagen," Arch Environ Health 32(2): 86-93, 1977.

176. Kerrebijn, J., et al., "Chronic Nonspecific Respiratory Disease in Children, A Five Year Follow-Up Study," Acta Paediatr Scand, Suppl. 261: 11-72, 1977.

177. Lebowitz, M. and B. Burrows, "Respiratory Symptoms Related to Smoking Habits of Family Adults," Chest 69(1): 48-50, 1976.

178. Love, G., et al., "The Incidence and Severity of Acute Respiratory Illness in Families Exposed to Different Levels of Air Pollution, New York Metropolitan Area, 1971-72," Arch Environ Health 36(2): 66-73, 1981.

179. Martin, C., et al., "Housing Conditions and Ill Health," Br Med J 294: 1125-1127, 1987.

180. Salzman, M.B., et al., "Passive Smoking and Croup," Arch Otolaryngol Head Neck Surg 113: 866-868, 1987.

2501457474

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

181. Shy, C., et al., "The Chattanooga School Children Study: Effects of Community Exposure to Nitrogen Dioxide. II. Incidence of Acute Respiratory Illness," J Air Pollut Control Assoc 20(9): 582-588, 1970.

182. Koo, L.C., et al., "A Comparison of the Prevalence of Respiratory Illnesses Among Nonsmoking Mothers and Their Children in Japan and Hong Kong," Am Rev Respir Dis 138: 290-295, 1988.

183. Melia, R., et al., "Association Between Gas Cooking and Respiratory Disease in Children," Br Med J II: 149-152, 1977.

184. Melia, R., et al., "Childhood Respiratory Illness and the Home Environment II. Association Between Respiratory Illness and Nitrogen Dioxide, Temperature and Relative Humidity," Int J Epidemiol 11(2): 164-169, 1982.

185. Melia, R.J.W., et al., "Respiratory Illness and Home Environment of Ethnic Groups," Br Med J 296: 1438-1441, 1988.

186. Melia, R., et al., "The Relation Between Respiratory Illness in Primary School Children and the Use of Gas for Cooking: I -- Results from a National Survey," Int J Epidemiol 8(4): 333-338, 1979.

187. Speizer, F. E., et al., "Respiratory Disease Rates and Pulmonary Function in Children Associated with NO_2 Exposure." Am Rev Respir Dis 121: 3-10, 1980.

188. Strachan, D. and R. Elton, "Relationship Between Respiratory Morbidity in Children and the Home Environment," Family Practice 3(3): 137-142, 1986.

189. Watkins, C., et al., "Patterns of Respiratory Illness in the First Year of Life," Br Med J 293: 794-796, 1986.

190. Kerigan, A., et al., "A Three-Year Cohort Study of the Role of Environmental Factors in the Respiratory Health of Children in Hamilton, Ontario: Epidemiologic Survey, Design, Methods, and Description of Cohort," Am Rev Respir Dis 133: 987-993, 1986.

191. Goren, A.I. and S. Hellmann, "Prevalence of Respiratory Symptoms and Diseases in Schoolchildren Living in a Polluted and in a Low Polluted Area in Israel," Environ Res 45(1): 28-37, 1988.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

192. Fleming, D., et al., "Childhood Upper Respiratory Tract Infections: To What Degree is Incidence Affected by Day Care Attendance?," Pediatrics 79(1): 55-60, 1987.

193. Berwick, M., et al., "Respiratory Illness in Children Exposed to Unvented Combustion Sources," Indoor Air: Radon, Passive Smoking, Particulates and Housing Epidemiology, eds. B. Berglund, et al. 1984: 255-260.

194. Berkey, C. S., et al., "Indoor Air Pollution and Pulmonary Function Growth in Preadolescent Children," Am J Epidemiol 123(2): 250-260, 1986.

195. Chen, Yue and W. X. Li, "The Effect of Passive Smoking on Children's Pulmonary Function in Shanghai," Am J Public Health 76(5): 515-518, 1986.

196. Corbo, G.M., et al., "Maternal Smoking and Bronchial Responsiveness in Children," Am Rev Respir Dis 137(1): 245, 1987.

197. Hasselblad, V., et al., "Indoor Environmental Determinants of Lung Function in Children," Am Rev Respir Dis 123(5): 479-485, 1981.

198. Tager, I., et al., "Effect of Cigarette Smoking on the Pulmonary Function of Children and Adolescents," Am Rev Respir Dis 131(5): 752-759, 1985.

199. Tager, I., et al., "Longitudinal Study of the Effects of Maternal Smoking on Pulmonary Function in Children," N Engl J Med 309(120): 699-703, 1983.

200. Tashkin, D., et al., "The UCLA Population Studies of Chronic Obstructive Respiratory Disease. VII. Relationship Between Parental Smoking and Children's Lung Function," Am Rev Respir Dis 129: 891-897, 1984.

201. Dodge, R., "The Effects of Indoor Pollution on Arizona Children," Arch Environ Health 37(3): 151-155, 1982.

202. Hosein, R. and P. Corey, "Multivariate Analysis of Nine Indoor Factors on FEV^1 of Caucasian Children", American Thoracic Society Meeting, Miami Beach, FL, May 20-23, 1984. Abstract.

203. Mandi, A., et al., "Relationship Between Lung Function Values and Air Pollution Data, in Budapest School Children," Pneumonologie 150(2/4): 217-225, 1974.

2501457476

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

204. Witorsch, R. and P. Witorsch, "Maternal Smoking and Pulmonary Performance in Children: A Critical Analysis," Indoor Air '87, Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Berlin, August 17-21, 1987: 175-179.

205. Lebowitz, M., et al., "Respiratory Symptoms and Peak Flow Associated with Indoor and Outdoor Air Pollutants in the Southwest," J Air Pollut Control Assoc 35: 1154-1158, 1985.

206. Justus, D. and D. Adams, "Evaluation of Tobacco Hypersensitivity Responses in the Mouse: A Potential Animal Model for Critical Study of Tobacco Allergy," Int Arch Allergy Appl Immunol 51(6): 687-695, 1976.

207. Lehrer, S., et al., "Immunochemical Properties of Tobacco Smoke and Leaf Components," Fed Proc 37(6): 1456, 1978.

208. Lehrer, S., et al., "Immunogenic Properties of Tobacco Smoke," J Allerg Clin Immunol 62(6): 368-370, 1978.

209. Taylor, G., "Tobacco Smoke Allergy -- Does It Exist?," Environmental Tobacco Smoke Effects on the Nonsmoker: Report From a Workshop, ed. R. Rylander, Scand J Respir Dis Suppl. 91: 50-55, 1974.

210. Lehrer, S., et al., "Immunogenicity of Tobacco Smoke Components in Rabbits and Mice," Int Arch Allergy Appl Immunol 62: 16-22, 1980.

211. Lehrer, S., et al., "Tobacco Smoke Sensitivity: A Result of Allergy?," Ann Allergy 56: 1-10, 1986.

212. Gleich, G., et al., "Allergy to Tobacco Antigens," J Allerg Clin Immunol 63(3): 211-212, 1979.

213. Harkavy, J., "Tobacco Allergy in Cardiovascular Disease: A Review," Ann Allerg 26: 447-459, 1968.

214. Speer, F., "Tobacco and the Nonsmoker: A Study of Subjective Symptoms," Arch Environ Health 16(3): 443-446, 1968.

215. Stankus, R.P. and S.B. Lehrer, "Inhalation Challenge Studies on Tobacco Smoke Sensitive Asthmatics," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 303-304.

2501457477

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

216. Stahle, I. and L. Tibbling, "Tobaksallergi hos Patienter Med Asthma Bronchiale (Tobacco Allergy Among Patients with Bronchial Asthma)," Lakartidningen 75(17): 1711-1713, 1978.

217. Tibbling, K., "Elicitation of Bronchial Asthma with Tobacco Extract," Abstracts of the 4th World Conference on Smoking and Health, Stockholm, Sweden, June 18-21, 1979.

218. Bylin, G., "Tobaksallergi -- finns den? (Tobacco Allergy -- Does It Exist?)," Lakartidningen 77(16): 1530-1532, 1980. Translation.

219. Salvaggio, J., Statement, U.S. Congress Committee on Agriculture, Subcommittee on Tobacco, Effect of Smoking on Nonsmokers, Hearing, 95th Cong., 2nd Sess., September 7, 1978. (Washington: Government Printing Office, 1978): 46-49.

220. McDougall, J. and G. Gleich, "Tobacco Allergy -- Fact or Fancy?," J Allerg Clin Immunol 57(3): 237, 1976. Abstract.

221. Lehrer, S., et al., "Immunological Evaluation of Tobacco Smoke 'Sensitive' Individuals by Skin, Radioallergosorbent and Precipitin Tests," J Allerg Clin Immunol 67: 49, 1981. Abstract.

222. Melius, J., et al., "Indoor Air Quality -- the NIOSH Experience," Ann Am Conf Gov Ind Hyg 10: 3-7, 1984.

223. Sterling, T. and E. Sterling, "Investigations on the Effect of Regulating Smoking on Levels on Indoor Pollution and on the Perception of Health and Comfort of Office Workers," ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels, eds. R. Rylander, et al., Eur J Respir Dis, Suppl. 133(65): 17-31, 1984.

224. Robertson, G., "Source, Nature and Symptomology on Indoor Air Pollutants," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk, (London: Selper Ltd., 1988): 311-319.

225. Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," J Occup Med 29(1): 57-62, 1987.

226. Pimm, P., et al., "Physiological Effects of Acute Passive Exposure to Cigarette Smoke," Arch Environ Health 33(4): 201-213, 1978.

227. Sterling, T., et al., "Indoor Byproduct Levels of Tobacco Smoke: A Critical Review of the Literature," J Air Pollut Control Assoc 32(3): 250-259, 1982.

2501457478

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

228. Nystrom, C. and C. Green, "Assessing the Impact of Environmental Tobacco Smoke on Indoor Air Quality: Current Status," Presentation, ASHRAE Indoor Air Quality Conference, Atlanta, GA, April 21, 1986.

229. Hoepfner, I., et al., "Hydroxy-Phenanthrenes in the Urine of Non-smokers and Smokers," Toxicology Letters 35(1): 67-71, 1987.

230. Scherer, G., et al., "Urinary Mutagenicity After Controlled Exposure to Environmental Tobacco Smoke (ETS)," Toxicology Letters 35(1): 135-140, 1987.

231. Sonnenfeld, G. and D. Wilson, "The Effect of Smoke Age and Dilution on the Cytotoxicity of Sidestream (Passive) Smoke," Toxicology Letters 35(1): 89-94, 1987.

232. Husgafvel-Pursiainen, K., "Sister-Chromatid Exchange and Cell Proliferation in Cultured Lymphocytes of Passively and Actively Smoking Restaurant Personnel," Mutation Research 190: 211-215, 1987.

233. Adlkofer, F., et al., "Exposure of Hamsters and Rats to Sidestream Smoke of Cigarettes: Preliminary Results of a 90-Day-Inhalation Study," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 252-258.

234. Aviado, D.M., "Suspected Pulmonary Carcinogens in Environmental Tobacco Smoke," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 141-146.

235. First, M., "Environmental Tobacco Smoke Measurements: Retrospect and Prospect," ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels, eds. R. Rylander, et al., Eur J Respir Dis, Suppl. 133(65): 9-16, 1984.

236. Jenkins, R. and M. Guerin, "Analytical Chemical Methods for the Detection of Environmental Tobacco Smoke Constituents," ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels, eds. R. Rylander, et al., Eur J Respir Dis, Suppl. 133(65): 33-46, 1984.

237. Russell, M., et al., "Absorption by Non-Smokers on Carbon Monoxide From Room Air Polluted by Tobacco Smoke," Lancet I: 576-579, 1973.

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

238. Harke, H., et al., "Zum Problem des Passivrauchens II. Untersuchungen uber den Kohlenmonoxidgehalt der Luft im Kraftfahrzeug durch das Rauchen von Zigaretten (The Problem of Passive Smoking. II. Investigations of CO Level in the Automobile After Cigarette Smoking)," Int Arch Arbeitsmed 33(3): 207-220, 1974. Translation.

239. Anderson, G. and T. Dalhamn, "Halsoriskerna vid passiv rökning (Health Hazards from Passive Smoking)," Lakartidningen 70: 2833-2836, 1973. Translation.

240. Bridge, D. and M. Corn, "Contribution to the Assessment of Exposure of Nonsmokers to Air Pollution From Cigarette and Cigar Smoke in Occupied Spaces," Environ Res 5(2): 192-209, 1972.

241. Duncan, D. and P. Greavey, "Passive Smoking and Uptake of Carbon Monoxide in Flight Attendants," JAMA 251(20): 120-21, 1984.

242. Hugod, C., "Passive Smoking," Ugeskr Laeger 143(34): 2181-2184, 1981. Translation.

243. Jones, R. and R. Fagan, "Carboxyhemoglobin in Nonsmokers: A Mathematical Model," Arch Environ Health 30(4): 184-189, 1975.

244. Klosterkötter, W. and E. Gono, "Zum Problem des Passivrauchens (The Problem of Passive Smoking)," Zbl Bakt Hyg, I abt Orig 162: 51-69, 1976. Translation.

245. Szadkowski, D., et al., "Kohlenmonoxidbelastung durch Passivrauchen in Büroräumen (Body Burden of Carbon Monoxide From Passive Smoking in Offices)," Inn Med 3(6): 310-313, 1976.

246. Kirk, P.W., et al., "Environmental Tobacco Smoke in Indoor Air," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 99-112.

247. Hutcheon, D., Statement, State of New Jersey Public Health Council, Public Hearing on Regulation of Smoking in Certain Public Places, Trenton, New Jersey, December 12, 1978.

248. Stewart, R., "The Effects of Low Concentrations of Carbon Monoxide in Man," Environmental Tobacco Smoke Effects on the Nonsmoker: Report from a Workshop, ed. R. Rylander, Scand J Respir Dis, Suppl. 91: 56-62, 1974.

2501457480

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

249. Wade, W., "A Study of Indoor Air Quality," J Air Pollut Control Assoc 25(9): 933-939, 1975.

250. Yocom, J., "Indoor-Outdoor Air Quality Relationships," J Air Pollut Control Assoc 32(5): 500-520, 1982.

251. Cox, B. D. and M. J. Whichelow, "Carbon Monoxide Levels in the Breath of Smokers and Nonsmokers: Effect of Domestic Heating Systems," J Epidemiol Community Health 39: 75-78, 1985.

252. Girman, J. and G. Traynor, "Indoor Concentrations," J Am Pollut Control Assoc 33(2): 89, 1983.

253. Repace, J. and A. Lowrey, "Indoor Air Pollution, Tobacco Smoke, and Public Health," Science 208: 464-472, 1980.

254. Weber, A. and T. Fischer, "Passive Smoking at Work," Int Arch Occup Environ Health 47: 209-221, 1980.

255. Gieseke, J., et al., "Collection and Characterization of Airborne Particulate Matter in Buildings," ASHRAE Transactions 84(1): 572-589, 1978.

256. Spengler, J., et al., "Long-Term Measurements of Respirable Sulfates and Particles Inside and Outside Homes," Atmos Environ 15: 23-30, 1981.

257. Press Release, "Study of Air Quality in 100 N.Y.C. Restaurants, Offices Shows Tobacco Smoke is Insignificant Factor," The (U.S.) Tobacco Institute, December 10, 1986.

258. IT Corporation, Final Report: Environmental Tobacco Smoke Survey, Dallas, Texas, Submitted to Tobacco Institute, Washington, D.C., August 4, 1987.

259. Carson, J.R. and C.A. Erikson, "Results from Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988.

260. Sterling, T., "ETS Concentrations Under Different Conditions of Ventilation and Smoking Regulation," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 89-98.

261. Ogden, M.W. and K.C. Maiolo, "Collection and Analysis of Solanesol as a Tracer of Environmental Tobacco Smoke (ETS)," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 77-88.

2501457481

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

262. Proctor, C.J., "The Analysis of the Contribution of ETS to Indoor Air," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 57-66.

263. Eatough, D.J., et al., "Assessing Exposure to Environmental Tobacco Smoke," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 131-140.

264. First, M., "Constituents of Sidestream and Mainstream Tobacco Smoke and Markers to Quantify Exposure to Them," Indoor Air and Human Health (Chelsea, Michigan: Lewis Publishers, 1985): 195-203.

265. Sterling, T., "Discussion -- Tobacco Smoke, Ventilation, and Indoor Air Quality," ASHRAE Transactions 88(1): 911-912, 1982.

266. Bouhuys, A., et al., "Do Present Levels of Air Pollution Outdoors Affect Respiratory Health?," Nature 276(5687): 466-471, 1978.

267. Badre, R., et al., "Pollution Atmospherique par la Fumee de Tabac (Atmospheric Pollution by Smoking)," Ann Pharm Fr 36(9-10): 443-452, 1978. Translation.

268. Harke, H., et al., "Passivrauchen: Abhängigkeit der Konzenstration von Rauchinhaltsstoffen in der Luft verschieden grosser Raumer von der Zahl der verrauchten Zigaretten und der Zeit (Passive Smoking: Concentration of Smoke Constituents in the Air of Large and Small Rooms as a Function of Number of Cigarettes Smoked and Time)," Int Arch Arbeitsmed 29(4): 323-339, 1972. Translation.

269. Hinds, W. and M. First, "Concentrations of Nicotine and Tobacco Smoke in Public Places," New Engl J Med 292(16): 844-845, 1975.

270. Jenkins, R.A., et al., "Development and Application of a Thermal Desorption-Based Method for the Determination of Nicotine in Indoor Environments," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 557-566.

271. Murumatsu, M., et al., "Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor," Environ Res 35: 218-227, 1984.

272. Muramatsu, M., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch Occup Environ Health 59: 545-550, 1987.

2501457482

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

273. Hinds, W., "The Lung and the Environment," Semin Respir Med 1(3): 197-210, 1980.

274. Harke, H. and A. Bleichert, "Zum Problem des Passivrauchens (On the Problem of Passive Smoking)," Int Arch Arbeitsmed 29: 312-333, 1972. Translation.

275. Ohkubo, C., "Some Acute Cardiopulmonary Effects of Mainstream and Sidestream Cigarette Smoke in Man," Prev Med 11: 173-186, 1982.

276. Foliart, D., et al., "Passive Absorption of Nicotine in Airline Flight Attendants," New Engl J Med 308(18): 1105, 1983.

277. Oldaker, G.B. and F.C. Conrad, "Estimation of Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Environ Sci Technol 21: 994-999, 1987.

278. Ember, L., "Nitrosamines: Assessing the Relative Risk," Chem Eng News 58(13): 20-26, 1980.

279. Lehnert, G., "Schlusswort: Zum Bereich des Spekulativen (Conclusion: In the Realm of Speculation)," Munch med Wschr 124(4): 19-20, 1982. Translation.

280. Schievelbein, H., "Are There Really Important New Findings About Passive Smoking?," Oeff Gesundheitswes 44(0): 454-456, 1982. Translation.

281. Brunnemann, K., et al., "Assessment of Carcinogenic Volatile N-Nitrosamines in Tobacco and in Mainstream and Sidestream Smoke from Cigarettes," Cancer Res 37: 3218-3222, 1977.

282. Brunnemann, K., et al., "The Influence of Tobacco Smoke on Indoor Atmospheres. II. Volatile and Tobacco Specific Nitrosamines in Main- and Sidestream Smoke and Their Contribution to Indoor Pollution," 4th Joint Conference on Sensing of Environmental Pollutants, 1977 (Washington, D.C.: American Chemical Society, 1978): 876-880.

283. Brunnemann, K. and D. Hoffmann, "Chemical Studies on Tobacco Smoke. LIX. Analysis of Volatile Nitrosamines in Tobacco Smoke and Polluted Indoor Environments," Environmental Aspects of N-Nitroso Compounds, eds. E. A. Walker, et al. (Lyon: IARC Scientific Publications, 1978): 343-355.

2501457483

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

284. Stehlik, G., et al., "Concentration of Dimethylnitrosamine in the Air of Smoke-Filled Rooms," Ecotoxicol Environ Safety 6: 495-500, 1982.

285. Good, B., et al., "Effect of Cigarette Smoking on Residential NO_2 Levels," Environ Intl 8: 167-175, 1982.

286. Hugod, C., et al., "Exposure of Passive Smokers to Tobacco Smoke Constituents," Int Arch Occup Environ Health 42: 21-29, 1978.

287. Holzer, G., et al., "Gas Chromatographic-Mass Spectrometric Evaluation of Exhaled Tobacco Smoke," J Chromatogr 126: 771-785, 1976.

288. Sega, K. and M. Fugas, "Nitrogen Dioxide Concentrations in Residences," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 493-496.

289. Bayer, C.W. and M.S. Black, "Thermal Desorption/Gas Chromatographic/Mass Spectrometric Analysis of Volatile Organic Compounds in the Offices of Smokers and Nonsmokers," Biomed Environ Mass Spect 14(8): 363-367, 1987.

290. Hollowell, C. and R. Miksch, "Sources and Concentrations of Organic Compounds in Indoor Environments," Bull NY Acad Med 57(10): 962-977, 1981.

291. Matsukura, S., et al., "Effects of Environmental Tobacco Smoke on Urinary Cotinine Excretion in Nonsmokers", New Engl J Med 311(13): 828-832, 1984.

292. Greenberg, Robert A., et al., "Measuring the Exposure of Infants to Tobacco Smoke," New Engl J Med 310(17), 1075-1078, 1984.

293. Hoffmann, Dietrich, et al., "Tobacco Sidestream Smoke: Uptake by Nonsmokers," Prev Med 13: 608-617, 1984.

294. Jarvis, M. J., et al., "Passive Exposure to Tobacco Smoke: Saliva Cotinine Concentrations in a Representative Population Sample of Non-Smoking School Children," Br Med J 291: 927-929, 1985.

295. Jarvis, Martin J. and Michael A. H. Russell, "Measurement and Estimation of Smoke Dosage to Non-Smokers From Environmental Tobacco Smoke," Environmental Tobacco Smoke Report from a Workshop on Effects and Exposure Levels, eds. R. Rylander, et al., Eur J. Respir Dis, Suppl. 133 (65): 68-75, 1984.

2501457484

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

296. Wald, Nicholas J., et al., "Urinary Cotinine as Marker of Breathing Other People's Tobacco Smoke," Lancet I, 230-231, 1984.

297. Jarvis, M., et al., "Passive Smoking in Adolescents: One-Year Stability of Exposure in the Home," The Lancet I: 1324-1325, 1987.

298. Benowitz, Neal L., "The Use of Biologic Fluid Samples In Assessing Tobacco Smoke Consumption," Measurement in the Analysis and Treatment of Smoking Behavior, U.S. National Institute on Drug Abuse Research Monograph 48: 6-25, 1983.

299. Benowitz, Neal L., et al., "Cotinine Disposition and Effects," Clin Pharmacol Ther 34(5): 604-611, 1983.

300. Johnson, L. C., et al., "Passive Smoking Under Controlled Conditions," Int Arch Occup Environ Health 56, 1985: pp. 99-110.

301. Letzel, H., et al., "Measuring Problems in Estimating the Exposure to Passive Smoking Using the Excretion of Cotinine," Toxicology Letters 35(1): 35-44, 1987.

302. Biber, A., et al., "Determination of Nicotine and Cotinine in Human Serum and Urine: An Interlaboratory Study," Toxicology Letters 35(1): 45-52, 1987.

303. Schwartz, S., et al., "Mathematical Modelling of Nicotine and Cotinine as Biological Markers of Environmental Tobacco Smoke Exposure," Toxicology Letters 35(1): 53-58, 1987.

304. Balter, N.J., et al., "Application of Physiological Pharmacokinetic Modeling to the Design of Human Exposure Studies with Environmental Tobacco Smoke," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 179-188.

305. Castro, A. and N. Monji, "Dietary Nicotine and Its Significance in Studies on Tobacco Smoking," Bio Arch, 2(2): 91-97, 1986.

306. Adlkofer, F., et al., "Passive Smoking," N Engl J Med 312(11): 719-720, 1984.

307. Pittenger, D., "Passive Smoking," N Engl J Med 312(11): 720, 1984.

2501457485

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

308. U.S. Civil Aeronautics Board, "Airline Consumer Complaints Down 31% in First Half of 1982," CAB News, September 20, 1982.

309. Cohen, R., Statement, U.S., Congress, House, Committee on Agriculture, Subcommittee on Tobacco, Effect of Smoking on Nonsmokers, Hearing, 95th Cong., 2nd Sess., September 7, 1978 (Washington: Government Printing Office, 1978): 69-75.

310. Response Analysis Corporation, Overall Report: Smoking and Productivity in the Work Place, April, 1984.

311. Feinhandler, S., Statement, U.S., Congress, House, Committee on Agriculture, Subcommittee on Tobacco, Effect of Smoking on Nonsmokers, Hearing, 95th Cong., 2nd Sess., September 7, 1978 (Washington: Government Printing Office, 1978): 56-63.

312. Lehrer, S., et al., "Tobacco Smoke 'Sensitivity' -- Is There an Immunologic Basis?," J Allergy Clin Immunol 73(2): 240-245, 1984.

313. Fettner, A., "Where There's Smoke There's Ire--The New 'Passive Smoking' Rules: Sidestepping Issues by Scapegoating Smokers," Voice 5-7, March 30, 1987.

314. Jones, W., "Inconclusive Evidence on the Harmful Effects of Smoking," U.S. Congressional Record - Extensions of Remarks, February 18, 1987: E489-E490.

315. Press Release, "Coalition of Labor Unions React to Surgeon General's Report on Smoking," (December 16, 1986).

316. Tobacco Institute, Tobacco Smoke and the Nonsmoker: Scientific Integrity at the Crossroads, (Washington, D.C.: The Tobacco Institute, 1986).

317. Ashe, R. and D. Vaughn, "Smoking in the Workplace: A Management Perspective," Emp Rel Law J 11(3): 383-406, 1985.

318. "Court Upholds Appeal Ruling on ETS," Tobacco Review 114(6): 12, 1987.

319. "Summary of Court Judgement Relating to Rights for Anti-Smoking," District Court (Tokyo, Japan) Ruling, 1-10, 1987.

2501457486

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000

320. U.S. Civil Aeronautics Board, "Smoking Aboard Aircraft, Final Rule," Federal Register 49(120): Part V, 14 CFR, Part 252, June 20, 1984.

321. Committee on Airliner Cabin Air Quality, Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council, The Airliner Cabin Environment: Air Quality and Safety, (Washington: National Academy Press, 1986).

322. U.S. Department of Transportation, Airline Cabin Air Quality: Report to Congress, (U.S. DOT, February, 1987).

323. U.S. Department of Transportation, Solicitation, Offer and Award, August 5, 1988.

324. Holcomb, L.C., "Impact of Environmental Tobacco Smoke on Airline Cabin Air Quality," Environ Technol Letters 9: 509-514, 1988.

325. WHO/IARC, Evaluation of the Carcinogenic Risk of Chemicals to Humans: Tobacco Smoking, IARC Monograph Series 38, Lyon, 1985.

326. Roe, F., "Passive Smoking", Lancet I: 888, 1988.

327. Zethraeus, S., "Arbetsskade - försäkringen är förmånlig men domen bevisor inte att passiv rökning ger cancer [Occupational Injury Insurance is Beneficial but the Judgment Does Not Prove that Passive Smoking Leads to Cancer"], Lag Avtal 1: 31-32, 1986. Translation.

328. "No Jobs If You Smoke: Bosses," The Daily (Sydney, Australia) Telegraph, July 22, 1988.

329. Press Release, "Sean Carroll Settlement," Tobacco Institute of Australia, July 21, 1988.

330. Crawford, W.A., "Health Effects of Passive Smoking in the Workplace," Indoor and Ambient Air Quality, eds. R. Perry and P.W. Kirk (London: Selper Ltd., 1988): 203-210.

331. Littlechild, S. and J. Wiseman, "Principles of Public Policy Relevant to Smoking," Policy Studies, Volume 4, Part 3, ed. Sir Charles Carter (London, Thetford Press, 1984): 1-14.

2501457487

Source: https://www.industrydocuments.ucsf.edu/docs/ktwk0000